ROLE OF MAGNETIC RESONANCE IMAGING IN CHRONIC PANCREATITIS

A THESIS SUBMITTED TO

BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT (PUNJAB)

IN THE PARTIAL FULFILLMENT

FOR THE AWARD OF DEGREE OF

DOCTOR OF MEDICINE

IN

RADIODIAGNOSIS

MAY 2022

Submitted By

DR. GAGANDEEP KAUR

DEPARTMENT OF RADIODIAGNOSIS
DAYANAND MEDICAL COLLEGE AND HOSPITAL
LUDHIANA, PUNJAB

DECLARATION BY THE CANDIDATE

I hereby declare that the thesis entitled "**ROLE OF MAGNETIC RESONANCE IMAGING IN CHRONIC PANCREATITIS**" submitted towards partial fulfillment of the requirements for the award of **MD in RADIODIAGNOSIS** degree to BFUHS is a compilation of original work carried out by me under the supervision of **Dr Chandan Kakkar**, Associate Professor, Department of Radiodiagnosis, **Dr. Ritu Dhawan Galhotra**, Professor, Department of Radiodignosis and **Dr. Ramit Mahajan**, Associate Professor, Department of Gastroenterology. No part of this thesis has formed the basis for the award of any degree previously.

_____

Dr. Gagandeep Kaur
Department of Radiodiagnosis,
Dayanand Medical College & Hospital,
Ludhiana, Punjab

Date:

Place:

CERTIFICATE OF SUPERVISORS

This is to certify that the work incorporated in this thesis entitled "**ROLE OF MAGNETIC RESONANCE IMAGING IN CHRONIC PANCREATITIS"** has been carried out by **Dr. Gagandeep Kaur** under our direct supervision. The data incorporated in this thesis is original and genuine and has been carried out by the candidate herself.

Signature of Supervisor

Dr. Chandan Kakkar
Associate Professor
Department of Radiodiagnosis
DMC&H, Ludhiana

Signature of Co-Supervisor

Dr. Ritu Dhawan Galhotra
Professor
Department of Radiodiagnosis
DMC&H, Ludhiana

Dr. Ramit Mahajan
Associate Professor
Department of Gastroenterology
DMC&H, Ludhiana

Date:

ENDORSEMENT BY THE HEAD OF THE INSTITUTE

This is to certify that the thesis entitled "**ROLE OF MAGNETIC RESONANCE IMAGING IN CHRONIC PANCREATITIS"** is a bonafide research work done by **Dr. Gagandeep Kaur** under the supervision of **Dr Chandan Kakkar**, Associate Professor, Department of Radiodiagnosis, **Dr. Ritu Dhawan Galhotra**, Professor, Department of Radiodignosis and **Dr. Ramit Mahajan**, Associate Professor, Department of Gastroenterology in this institution.

Principal

Dr. Sandeep Puri
Principal
DMC&H, Ludhiana

Date:

COPYRIGHT

Declaration by the Candidate

I hereby declare that the Baba Farid University of Heath Sciences, Faridkot, shall have the rights to preserve & use this thesis in print or electronic format for academic/study purpose by the research scholar/students.

Signature

Dr Gagandeep Kaur
Department of Radiodiagnosis,
Dayanand Medical College & Hospital,
Ludhiana, Punjab - 141001

Date:

© Baba Farid University of Health Sciences, Faridkot

Dedicated

to

my Family

&

Teachers

ACKNOWLEDGEMENT

It is not a mere formality to write an acknowledgement but it is my sacred duty to pay my debt of gratitude from the depth of my heart to my teachers, who have encouraged, inspired and guided me at every step of my work, without which my task would have been undone.

Words are insufficient to express my gratitude with great reverence to my noble guide Dr. Chandan Kakkar Associate Professor, Department of Radiodiagnosis, DMC Ludhiana, who not only served as my supervisor, but also encouraged and advised me throughout the study. All the constant support, departmental facilities, indispensable suggestions, constructive criticism, & timely help he has given to me over the past two and half years, has brought this work of mine to its present form. I wish him continued success and happiness in future.

I wish to express my profound gratitude to my Co-Guide Dr. Ritu D Galhotra, Professor, Department of Radiodiagnosis, DMC Ludhiana, for her valuable contribution, guidance, advice, potential attention & encouragement throughout the venture.

I express my thanks to my Co-Guide Dr. Ramit Mahajan, Associate Professor, Department of Gastroenterology, DMC Ludhiana for his moral support, useful criticism & caring attitude throughout this journey.

My sincere thanks to Dr. Kavita Saggar, Professor & HOD, Department of Radiodiagnosis, DMC Ludhiana for her invaluable contribution, constant help, advice & continuous inspiration that led to the successful completion of this work.

I am also highly indebted to my respected teachers Dr. Archana Ahluwalia, Professor, Dr. Siddharth Parkash Professor, Dr. Kamini Gupta, Professor, Dr. Devinderpal singh Dhanota, Assistant Professor, Dr. Satwant Singh Khela, Assistant Professor, Department of Radiodiagnosis, DMC Ludhiana whose encouragement and affection has always been a guiding light in my work.

I would like to thank my parents, my husband Dr. Mohit Mehmi, my seniors, batch-mates, friends, entire technical and official staff of Department of Radiodiagnosis, DMC who provided me all the valuable help, guidance, emotional support and patience.

I am extremely thankful to all the patients and volunteers who participated in this study. This project would have been unthinkable without their contribution.

No words could express my feelings of acknowledgement for the efforts put on by my parents in my academic pursuit. Without their moral support and help I would not be there where I am today.

Thank you almighty God for everything & for all the blessings.

Dr. Gagandeep Kaur

ABBREVIATIONS

SMA	SUPERIOR MESENTERIC ARTERY
GDA	GASTRODUODENAL ARTERY
MRI	MAGNETIC RESONANCE IMAGING
CT	COMPUTED TOMOGRAPHY
ERCP	ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY
EUS	ENDOSCOPIC ULTRASOUND
CP	CHRONIC PANCREATITIS
SE	STRAIN ELASTOGRAPHY
SWE	SHEAR WAVE ELASTOGRAPHY
GRE	GRADIENT ECHO SEQUENCES
SI	SIGNAL INTENSITY
SIR(P/L)-1	SIGNAL INTENSITY RATIO OF PANCREAS TO LIVER
SIR(P/S)-1	SIGNAL INTENSITY RATIO OF PANCREAS TO SPLEEN
SIR(P/PSM)-1	SIGNAL INTENSITY RATIO OF PANCREAS TO PARASPINAL MUSCLES
ROI	REGION OF INTEREST
SPSS	STATISTICAL PACKAGE FOR THE SOCIAL SCIENCE
ROC	RECEIVER OPERATING CHARACTERISTIC CURVE

ABSTRACT

Introduction- Chronic pancreatitis(CP) is characterized by continued inflammation and destruction that leads to irreversible morphological changes in the pancreatic parenchyma & its ductal anatomy. It is widely acknowledged that MRI with MRCP is more sensitive than is CT for detection of ductal and subtle parenchymal changes, especially during early stages of chronic pancreatitis. MRCP provides information on gland morphology, ductal morphology and signal changes in chronic pancreatitis which is a cornerstone of noninvasive assessment of the disease.

Objectives-To study the morphological, ductal and signal intensity pattern changes of pancreas in patients with chronic pancreatitis.

Material and methods-Prospective study was performed on a cohort of 90 patients (45 cases and 45 controls) for imaging assessment for chronic pancreatitis. MRCP was performed on patients with clinical and biochemical evidence of chronic pancreatitis. Signal pattern of pancreas (T1 fat saturated sequences), ductal changes and other morphological changes were assessed. Signal intensity ratio of pancreas with respect to liver, spleen and muscle were derived.

Results- Lowered T1 signal intensity (SI) was observed in 87% of patients with Chronic Pancreatitis on qualitative as well as quantitative analysis. Out of 45 cases, 13% showed a similar signal intensity of the pancreas as compared to the signal intensity of the liver. 0ut of 45 controls, 100% showed the higher signal intensity of the pancreas in comparison to other organs. Signal changes (sensitivity 86.67% & specificity 100 %) serve as a more sensitive parameter for chronic pancreatitis than ductal changes(sensitivity 82.2% & specificity 100%)

Conclusion- Visual analysis (Qualitative) of the pancreatic signal pattern is a dependable parameter for distinguishing normal from the abnormal pancreas. Quantitative analysis using signal intensity patterns has comparable performance. The pancreas–liver ratio is the best quantitative means of distinguishing normal from the abnormal pancreas. Signal changes (sensitivity 86.67% & specificity 100 %) serve as a more sensitive parameter for chronic pancreatitis than ductal changes (sensitivity 82.2% & specificity 100%) for early diagnosis of chronic pancreatitis.

TABLE OF CONTENTS

S. NO.	TOPIC	PAGES
I	INTRODUCTION	1-16
II	AIMS AND OBJECTIVES	17
III	REVIEW OF LITERATURE	18-24
IV	MATERIALS AND METHODS	25-31
V	OBSERVATIONS AND RESULTS	32-52
VI	DISCUSSION	53-60
VII	SUMMARY AND CONCLUSIONS	61-63
VIII	BIBLIOGRAPHY	64-70
	APPENDICES – I - PROFORMA - MASTER CHART	
	APPENDICES – II - APPROVAL LETTER OF THESIS PLAN - THESIS PLAN	

LIST OF TABLES

Table No.	Title	Page No.
1.	Cambridge Classification	30
2.	Mean age value among cases and controls	32
3.	Age distribution among cases and controls	33
4.	Gender distribution among cases and controls	34
5.	Distribution of patients according to Clinical history	35
6.	Biochemical parameters among cases and controls	36
7.	Mean parenchymal thickness among cases and controls	37
8.	Pancreatic parenchymal T1 SI	38
9.	Signal intensity ratios of pancreas to liver, spleen and paraspinal muscles	39
10.	ROC curves for quantitative assessment of the pancreas using liver as comparison tissue in the entire study population(i.e. SIR(P/L)-1, SIR(P/S)-1, SIR (P/PSM)-1)	41
11.	Pancreatic duct contour among cases and controls	42
12.	Pancreatic ductal side branches among cases and controls	43
13.	Severity of ductal changes among cases and controls	44
14.	Intraductal stricture among cases and controls	45
15.	Intraductal calculus among cases and controls	46
16.	Only T1 signal changes among cases and controls	47
17.	Both signal as well as ductal changes among cases and controls	49
18.	Percentage of cases and controls showing signal intensity changes	50
19.	Sensitivity and specificity of signal intensity changes among cases and controls	50
20.	Percentage of ductal changes in cases and controls	51
21.	Sensitivity and specificity of both signal intensity changes and ductal changes	52

LIST OF FIGURES

Fig. No.	Title	Page No.
1.	Illustration of the normal pancreatic anatomy.	2
2.	Anatomic relationships of the pancreas with surrounding organs and structures	3
3A	Development of pancreas	5
3B.	Development of pancreas	6
4	Anatomic variations in the pancreatic and common bile duct systems.	7
5	Haste T2 axial image shows normal pancreatic duct	15
6.	Axial T1-weighted fat-suppressed spoiled gradient-echo image shows normal signal intensity of pancreas	16
7.	Mean age value among cases and controls	32
8.	Age distribution among cases and controls	33
9.	Gender distribution among cases and controls	34
10.	Distribution of patients according to Clinical history	35
11.	Biochemical parameters among cases and controls	36
12.	Mean parenchymal thickness among cases and controls	37
13.	T1 signal intensities of pancreas, liver, spleen and paraspinal muscles	38
14.	Signal intensity ratios of pancreas to liver, spleen and paraspinal muscles	39
15.	Graph shows ROC curves for quantitative assessment of the pancreas using liver as comparison tissue in the entire study population	40
16.	Pancreatic duct contour among cases and controls	42
17.	Pancreatic ductal side branches among cases and controls	43
18.	Severity of ductal changes among cases and controls	44
19.	Intraductal stricture among cases and controls	45

20.	Intraductal calculus among cases and controls	46
21.	Only T1 signal changes among cases and controls	48
22.	Both signal as well as ductal changes among cases and controls	49
23.	Sensitivity and specificity of ductal changes in cases and controls	51

LIST OF PICTURES

Pic No.	Title
1.	Image representing no; 11 as in the master chart
2.	Image representing no; 11 as in the master chart
3.	Image representing no; 23 as in the master chart
4.	Image representing no; 23 as in the master chart
5.	Image representing no; 35 as in the master chart
6.	Image representing no; 35 as in the master chart
7.	Image representing no; 24 as in the master chart
8, 9	Image representing no; 24 as in the master chart
10.	Image representing no; 14 as in the master chart
11.	Image representing no; 14 as in the master chart
12.	Image representing no; 75 as in the master chart
13.	Image representing no; 75 as in the master chart
14, 15	Image representing no; 49 as in the master chart
16.	Image representing no; 49 as in the master chart

INTRODUCTION

GROSS ANATOMY

The pancreas is a retroperitoneal organ located in the anterior pararenal space posterior to the stomach and bounded by the C-loop of the duodenum on the right side. The pancreas is divided into the head, uncinate process, neck, body, and tail. The head is situated within the duodenal C-loop, while the tail lies in the splenic hilum slightly superior to the head. Since the pancreas is unencapsulated, there is the uninhibited spread of tumour and inflammation to surrounding structures.[1]

The splenic-portal vein confluence lies just posterior to the pancreatic neck and anterior to the uncinate process. The splenic vein courses along the posterior aspect of the pancreatic body and tail, while the celiac artery is related cranially to the neck of the pancreas. The superior mesenteric artery (SMA) arises from the aorta at the level of the pancreatic neck. The main pancreatic duct of Wirsung arises from the pancreatic tail and drains the tail, body, neck, and uncinate process. The main duct joins the common bile duct in the pancreatic head before draining into the major papilla of the duodenum. The accessory duct of Santorini mainly drains the head and drains in the minor papilla The head receives arterial blood supply from the common hepatic artery via gastroduodenal artery (GDA) branches—the anterior and posterior superior pancreaticoduodenal branches and the SMA-inferior pancreaticoduodenal branches. These form an extensive arterial network around the pancreatic head. The body and tail of the pancreas are supplied by branches of the splenic artery, including the dorsal pancreatic artery.[2]

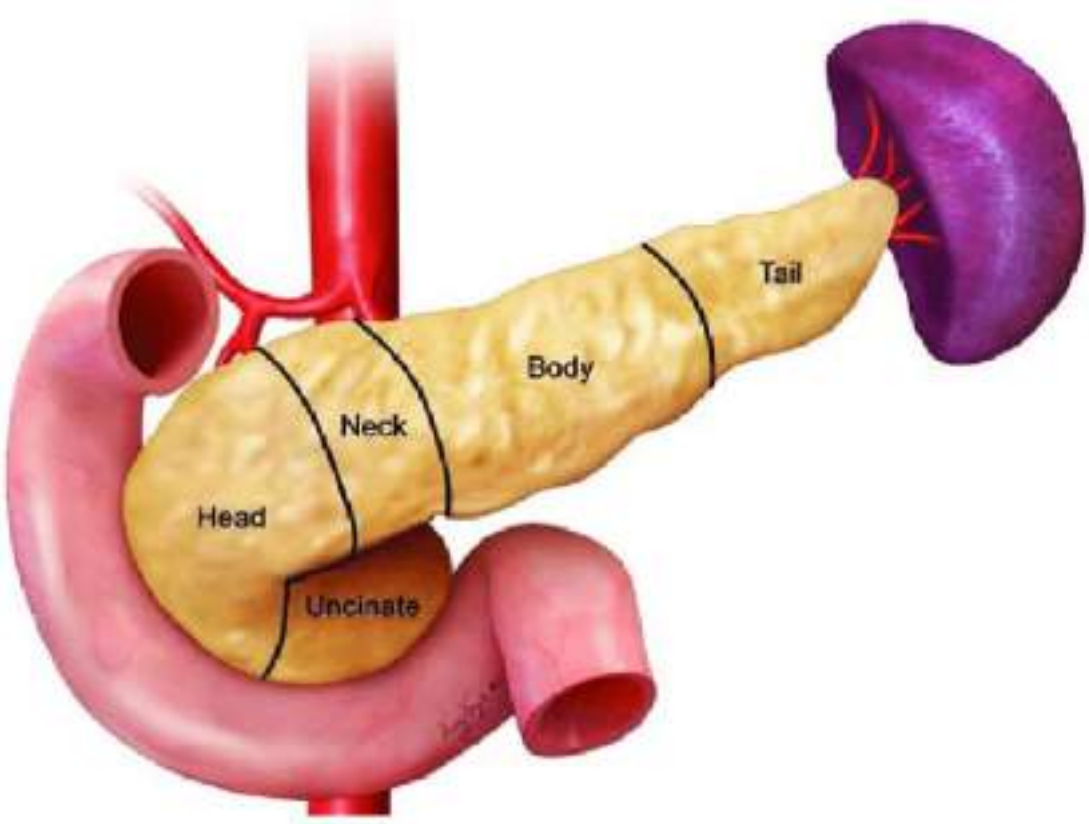


Fig 1. Illustration of the normal pancreatic anatomy. The pancreas is divided into head, neck, body, and tail. The uncinate process is a triangular prolongation of the caudal part of the head behind the superior mesenteric vessels. Image courtesy: Kelly Kage, Media Division, The University of Texas MD Anderson Cancer centre.

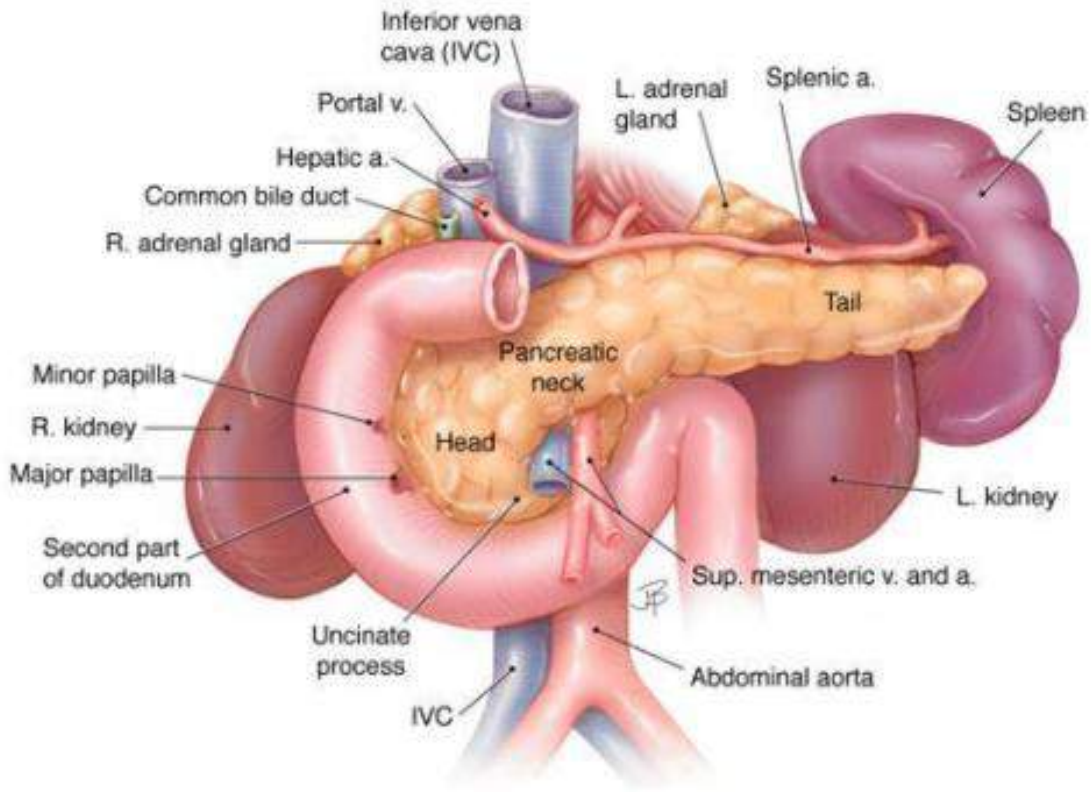


Figure 2. Anatomic relationships of the pancreas with surrounding organs and structures. Artwork in Figures 2. is by Jennifer Parsons Brumbaugh. These drawings were originally published in the AFIP Fascicle on pancreatic neoplasms and are used with permission of the publisher.

On T1-weighted sequences without fat saturation, the normal pancreas is isointense to slightly hyperintense compared with the liver. With fat-saturation, the pancreas appears hyperintense on T1-weighted imaging compared with the liver as a result of the T1 shortening effect of acinar protein within the normal pancreas. The fat-saturated sequence helps in suppressing the retroperitoneal fat and thus improves the contrast between the hyperintense pancreas and the fat-suppressed retroperitoneum.[3]

On T1-weighted postgadolinium imaging, the normal pancreas enhances homogenously and becomes isointense to the liver during the portal-venous phase. The normal pancreas is isointense to the liver and hypointense to the spleen on T2-weighted acquisitions.[4]

Heavily T2-weighted sequences, the signal of static or slow-moving fluid-filled structures such as the bile and pancreatic ducts is greatly increased, resulting in increased duct-to-background contrast. The combination of rapid sequences and the torso phased-array coil, which increases the signal-to-noise ratio, makes it possible to visualize ducts as small as 1 mm in diameter.
The coronal plane is used to provide a cholangiographic display, and the axial plane is used to evaluate the pancreatic duct and distal common bile duct.

The pancreatic duct is curved and obliquely oriented, it is usually not seen in its entirety on a single source image; however, the entire length can usually be evaluated when reviewing sequential images. Although an image with thick collimation (2–3 cm) can demonstrate the duct in its entirety, overlap of ducts may mimic a pseudocyst and volume averaging with adjacent fluid-filled intestine may obscure portions of the duct. Therefore, the multisection, thin-collimation images should be closely scrutinized. When the single-shot fast spin-echo technique is used, the main pancreatic duct in the head, body, and tail can be seen in up to 97%, 97%, and 83% of cases, respectively. However, because of a decrease in spatial resolution, the pancreatic side branches are seen less frequently, with secondary branches in the head, body, and tail seen in 19%, 10%, and 5% of cases, respectively.[5]

Normal anatomy on MRCP. The confluence of the right and left intrahepatic ducts to form the common hepatic duct is seen (*long thin arrow*). The cystic duct (*) typically joins the right side of the common hepatic duct to form the common bile duct (CBD) (*short arrow*). The main pancreatic duct (*arrowheads*) drains along with the CBD into the major duodenal papilla. An accessory pancreatic duct is present (*black circle*), draining into the minor duodenal papilla. Fluid containing structures such as the gallbladder (*GB*), duodenum (*D*) and stomach (*S*) is well seen on

this T2-weighted sequence with the duodenum obscuring part of the biliary tree.

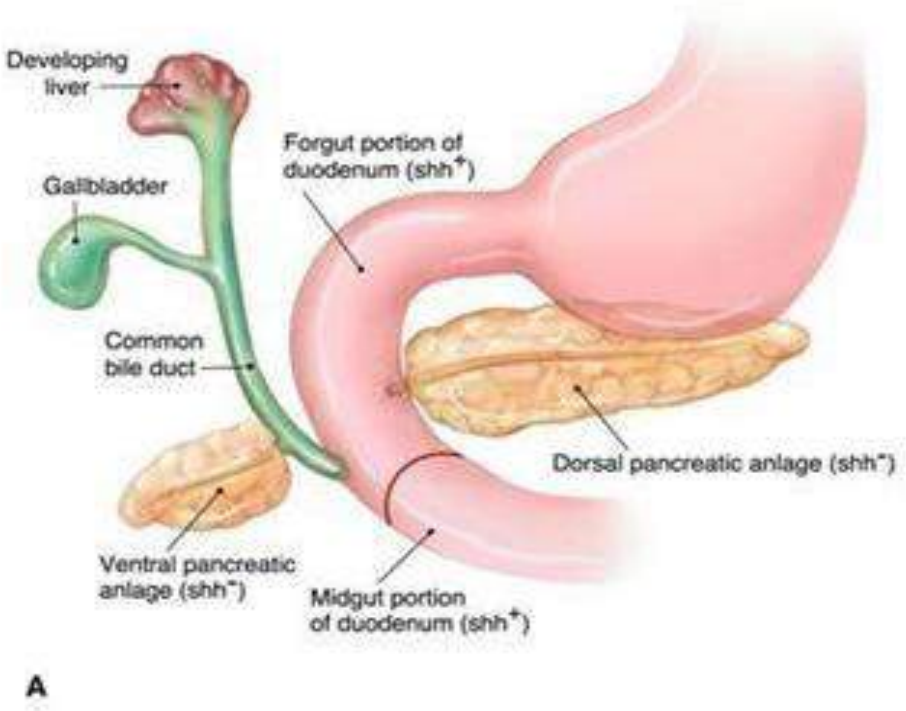


Fig 3(**A**) Development of pancreas: The pancreas develops from two outgrowths of the foregut distal to the stomach. The ventral diverticulum gives rise to the common bile duct, gallbladder, liver and the ventral pancreatic anlage that becomes a portion of the head of the pancreas with its duct system including the uncinate portion of the pancreas. The dorsal pancreatic anlage gives rise to a portion of the head, the body, and tail of the pancreas including a major duct that is continuous through the three regions. (Image by Jennifer Parsons Brumbaugh; used with permission of the publisher

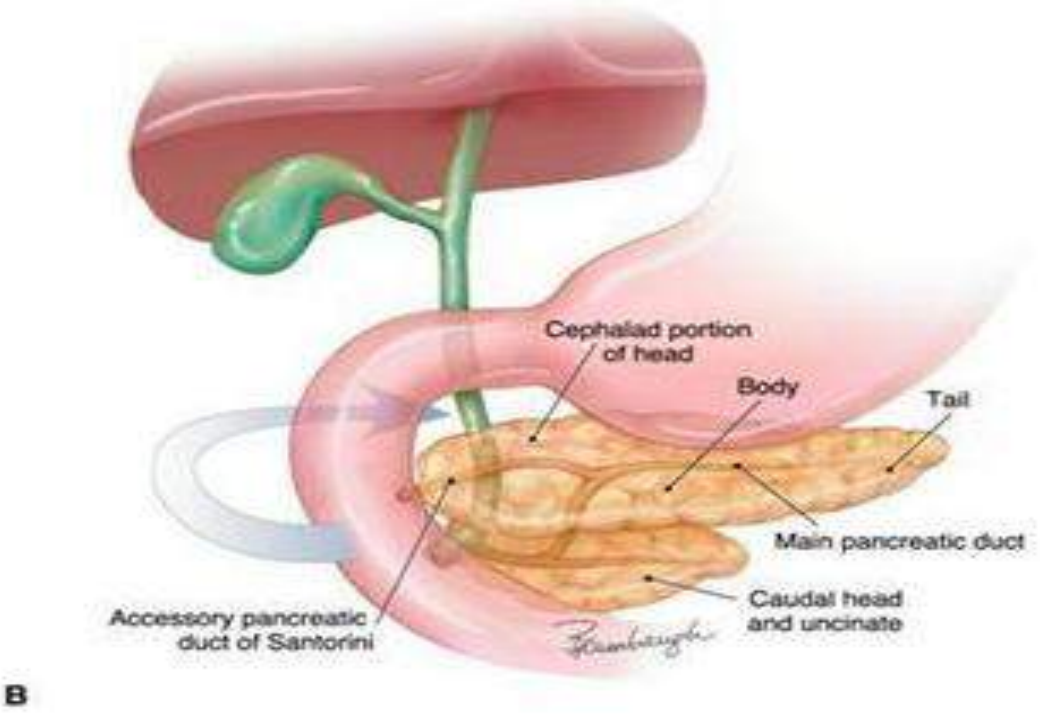


Fig 3: (**B**) The caudal portion of the head of the pancreas (uncinate) and the major papilla (ampulla of Vater) are derived from the ventral anlage. The minor papilla that drains the duct of Santorini is derived from the dorsal anlage. (Image by Jennifer Parsons Brumbaugh; used with permission of the publisher.

The course of the pancreatic duct varies greatly and the most common one (50%) is a descending course. Other courses include sigmoid, vertical, and loop configurations.

The ductal configuration most commonly (60%) manifests as a bifid configuration with a dominant duct of Wirsung. Less common configurations include an absent duct of Santorini (30%), a dominant duct of Santorini without divisum (1%), and 'ansa pancreatica', in which the duct of Santorini forms a reversed-S shape and connects with a side branch of the duct of Wirsung.

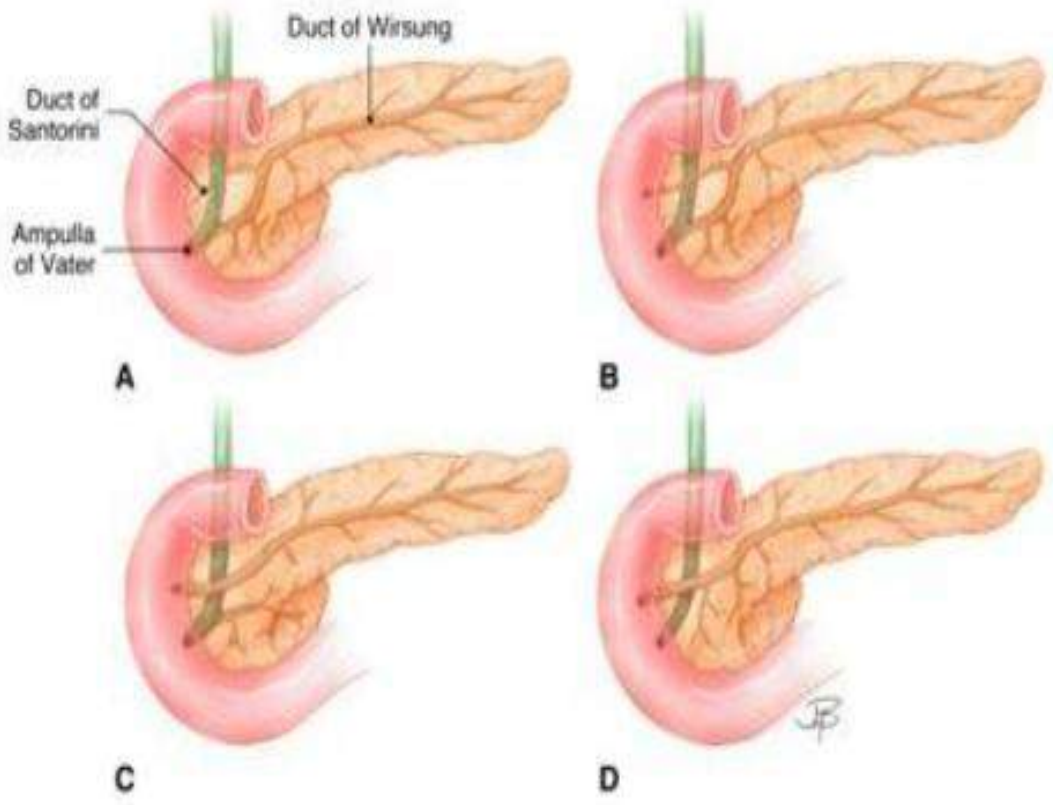


Fig 4: Anatomic variations in the pancreatic and common bile duct systems. The anatomic variations depicted provide additional examples of individual differences in pancreatic anatomy seen in adults. It becomes apparent that the duct of Santorini is derived from the dorsal anlage, whereas the duct of Wirsung (the main duct of the pancreas) is derived from the fusion of duct systems of both dorsal and ventral anlagen and drains into the duodenum at the ampulla of Vater as depicted in **A** and **B**. The connection of the duct of Santorini to the duodenum may regress as depicted in **A** or persist as in **B**, **C**, and **D**. The duct systems of the two anlagen may fail to fuse as depicted in **C** giving rise to “pancreas divisum”. Rarely the duct systems may fuse but lose their connection to the ampulla as depicted in **D**. Pancreatic secretions then reach the duodenum through the duct of Santorini and the minor papilla. (Image by Jennifer Parsons Brumbaugh; used with permission of the publisher.

Cystic dilatations of terminal portions of the ducts of Wirsung and Santorini are termed Wirsungocele and Santorinicele.[6]

Anomalous union of the pancreatic and bile ducts is seen when the junction of the pancreatic and common bile duct is outside of the duodenal wall, resulting in a combined channel leading to the major papilla that measures more than 1.5 cm in length. Therefore, the contraction of the sphincter of Oddi does not affect the pancreaticobiliary junction and causes pancreaticobiliary reflux, which may result in various clinical presentations such as pancreatitis.[7]

Pancreatic divisum, is characterized not only by the anatomical morphology but also by the physiology in which the majority of pancreatic juice drains through the duct of Santorini into the duodenum at the orifice of minor papilla while the minority (about 10%) drains through the (ventral) duct of Wirsung into the duodenum at major papilla.[8]

There are three major types of pancreatic divisum. Type I, or classic pancreatic divisum, is a complete failure of the dorsal and ventral buds to fuse. Type II pancreatic divisum is characterized by the absence of the ventral duct, so the minor papilla drains the entire pancreas and the major papilla drains some of the common bile ducts. Finally, type III presents with a small remnant communication between the dorsal duct and ventral duct.[9]

Although the clinical significance remains controversial, there seems to be an association between PD and chronic abdominal pain and recurrent acute pancreatitis. Moreover, the timely and appropriate therapeutic interventions such as minor papillotomy or stent placement in the dorsal pancreatic duct or surgical procedures can benefit the patients with symptomatic PD remarkably from reducing the pressure in the main pancreatic duct.[10]

CHRONIC PANCREATITIS

Chronic pancreatitis (CP) is an important gastrointestinal cause of morbidity worldwide. It can severely impair the quality of life besides life-

threatening acute and long-term complications. Pain and pancreatic exocrine insufficiency (leading to malnutrition) impact the quality of life. Acute complications include pseudocysts, pancreatic ascites, and vascular complications. Long-term complications are diabetes mellitus and pancreatic cancer. Early diagnosis of CP is crucial to alter the natural course of the disease.[11]

CP due to alcoholism constitutes about 70-90% of cases in western countries. Malnutrition is a rare cause of CP now.[12]

Other important risk factors are genetic predisposition, smoking, high protein diet, hypercalcemia, hyperparathyroidism, congenital lesions like abnormal pancreaticobiliary junction and pancreatic divisum, pancreatic or periampullary neoplasms, and ampullary stricture. Rarer causes of CP include hereditary pancreatitis, autoimmune pancreatitis, and chronic renal failure.[13]

Gall stones are the major cause of acute pancreatitis. However, they are not considered as risk factors for chronic pancreatitis.[14]

Early diagnosis of CP is difficult. Biochemical studies do not help in definitive diagnosis in the early stages

Diagnosis at this stage is suspected based on a combination of clinical symptoms, pancreatic function tests, and radiological investigations.[15]

Definitive diagnosis of CP is established in advanced cases with the destruction of greater than 90% of parenchyma.[16]

Several imaging modalities have been used to assess the pancreas such as abdominal radiographs, ultrasonography, computed tomography (CT), magnetic resonance imaging (MRI), and endoscopic retrograde cholangiopancreatography (ERCP). In the early phases of CP, functional and morphological disturbances can be seen at the ductal level, with the delayed discharge of pancreatic enzymes in response to secretin (functional) or ectasia and irregularities of the main/branch ducts (morphological).[17]

Imaging modalities

Radiographs**:** A radiograph is now an obsolete tool for the diagnosis of CP. The utility of radiographs in CP is restricted to the detection of focal calcifications along the course of the pancreas, in the epigastric region. Calcifications in CP is the most specific feature, however, it occurs late in the course of the disease. Radiographs have poor sensitivity in the diagnosis of CP.[18]

Ultrasound -ultrasound is easily available, cheap, and has no risk of ionizing radiation, it is used as the first-line tool in the radiological assessment of the pancreas.[19]

The pancreas should always be assessed in fasting status as this can restrict the obscuration of the gland by bowel gases. Graded compression can help to displace the bowel gases and result in better visualization of the pancreas. The pancreas is usually iso/hyperechoic compared with normal hepatic parenchyma. In the early stages of CP, ultrasound is seldom useful.[20]

In the later stages of the disease, there are focal hypoechoic areas and hyperechoic areas within the gland representing inflammatory and fibrotic tissues, respectively. Hence, CP gives a heterogenous appearance to the pancreas. Pancreatic parenchyma shows progressive atrophy in CP. Irregular progressive dilatation of the main and the branch pancreatic ducts are seen over time. Discontinuous strictures within the duct may give a “chain of lakes” appearance. Punctate calcifications with or without posterior acoustic shadowing, in the pancreatic parenchyma and the ducts, is the hallmark of CP, seen in about 40% of cases. Tiny calcifications not seen on routine sonograms can be identified with colour Doppler as a result of twinkling artefacts.[21]

Other important findings in CP that can be detected with sonography are pseudocysts, thrombosis of the splenoportal axis, and portosystemic collaterals and arterial complications like aneurysms most commonly of the splenic artery or gastroduodenal artery.[22]

Endoscopic Ultrasonography (EUS)

Transabdominal sonography has a limited scope in the assessment of the pancreas in obese individuals and gaseous abdomen. This limitation is overcome by EUS, as the high-frequency transducer (5-12.5 MHz) in EUS is close to the pancreas, hence visualizing it better.[23]

Normal pancreas in EUS shows homogenous appearance, smooth margins, and is slightly hyperechoic to the liver (salt pepper appearance). The normal main pancreatic duct is tubular with anechoic walls. Normal side branches can be seen in about 32% of patients.[24,25]

Certain features of CP seen in EUS besides the usual features seen in TAS and CT are subtle lobularity of the gland borders, tiny cystic changes within the glandular parenchyma, echogenic foci/strands representing areas of fibrosis, side branch ectasia, and echogenic margins of the main pancreatic duct. EUS due to its superior resolution compared with TAS and CT helps in the early diagnosis of CP by detecting the subtle parenchymal and ductal changes.[26]

Elastography

Elastography is a non-invasive ultrasound technique that helps to assess the stiffness of tissue. Elastography can be qualitative or quantitative. The stiffness of the region of interest (ROI) can be quantified either by conventional strain elastography (SE) or by shear wave elastography (SWE)

As the first generation strain elastography was qualitative, second-generation systems were developed, which gave the strain ratio (SR). ROIs were drawn over the target region and the surrounding reference region (preferably in the gut wall). Then the SR was calculated.[27,28]

SWE quantitatively determines the stiffness of the tissue based on the velocity of shear waves in the tissues. Both SWE and SE yield elastograms, which are colour maps superimposed on the grey-scale images. The colour pattern determines the degree of stiffness (blue being towards the hard end of the spectrum and red towards the soft end).[29]

In CP, fibrosis of the gland leads to a progressive increase in glandular stiffness.[30]

Focal pancreatic lesions can also be evaluated with EUS elastography to determine the nature based on the strain pattern.[31]

With the progression of the disease, there is a corresponding decline in the exocrine activity of the gland. EUS elastography helps in predicting the enzymatic deficiency, suggesting the requirement of enzyme replacement therapy in CP.[32]

ERCP

ERCP which was once considered as the gold standard test for CP is seldom used for diagnosis of CP at present. ERCP and magnetic resonance cholangiopancreatography (MRCP) can complement CT by better visualizing the pancreatic ductal system. Before the widespread use of magnetic resonance imaging, ERCP was the gold standard to confirm early chronic pancreatitis. ERCP sensitivity and specificity for the diagnosis of chronic pancreatitis has been reported to be 70% to 90%, and 90% to 100%, respectively. Currently, ERCP is not routinely performed purely for diagnostic purposes because it is invasive and can induce acute pancreatitis; its use is largely restricted to situations where endoscopic therapy is contemplated, such as for stone removal MRCP is now preferred for diagnosis of CP. Agreement of 83–100% for ductal dilatation, 70–92% for identification of strictures, and 92–100% for identification of filling defects has been revealed on comparison of MRCP and ERCP.[33]

Computed tomography

Calcifications are seen in the late stages of CP and hence detection of CP in early stages using CT is difficult. The sensitivity of CT in detecting late stages of CP is 60- 95%.[34]

Alcohol-induced pancreatitis and hereditary pancreatitis show calcifications in the gland at a relatively earlier stage than in other causes of pancreatitis like obstructive CP and cystic fibrosis-related CP.[35]

The presence and number of parenchymal calcifications is independent predictor of the degree of pancreatic fibrosis.[36]

The multiphase protocol is now commonly used in the assessment of the pancreas, which includes a pre-contrast scan which helps in the excellent depiction of calcifications, a pancreatic phase which is the best phase to assess the arterial enhancement for surgical mapping and to study the arterial complications, followed by a venous phase which shows enhancement of the pancreatic parenchyma. This phase is best suited to evaluate the parenchyma, pancreatic duct, focal lesions, and collections MRI/MRCP has superseded CT in the detection of pancreatic ductal abnormalities, however, CT is still the preferred modality to assess the complications of CP.[37]

Magnetic Resonance Imaging (MRI)

Cross-sectional imaging is the most commonly used method to establish a diagnosis of acute pancreatitis and CP. Among cross-sectional imaging studies, CT is readily available and is performed often in the setting of acute pancreatitis. It is widely acknowledged that MRI with MR cholangiopancreatography is more sensitive than is CT for detection of ductal and subtle parenchymal changes, especially during early stages of CP.[38]

Imaging provides comprehensive information about pancreatic composition and volume, ductal changes, and calcifications. Serial imaging is well suited as a noninvasive test for longitudinal monitoring. There is currently no imaging classification for diagnosis and disease severity that incorporates parenchymal and ductal abnormalities. Studies evaluating the T1 signal of the pancreas demonstrated correlation with exocrine insufficiency of the pancreas.[39]

T1 mapping, extracellular volume fraction, and diffusion-weighted imaging are useful quantitative imaging parameters for diagnosis and severity of CP.[40] First, instead of calcifications (a feature detected by using CT with high sensitivity), we will focus on the detection of parenchymal fibrosis.

Based on the literature, parenchymal T1 signal intensity changes and parenchymal enhancement pattern during the arterial and venous phases are features that have been shown to reflect tissue inflammation and/ or fibrosis.[41] Second, ductal morphology is far better evaluated with MR cholangiopancreatography than with CT, especially with administration of secretin. Hence, elements from the Cambridge classification have been adopted to score ductal morphology.[42] Reporting of MRI or MR cholangiopancreatography incorporates both ductal and parenchymal findings. Similar to CT, MR findings include both subjective (eg, duct contour irregularity) and objective (quantitative) measurements of the parenchyma (eg, T1 signal intensity and arteriovenous enhancement ratio). Various pulse sequences and approaches have been described for imaging the pancreatic duct and biliary tract with MRCP.[43] Imaging the pancreatic duct is dependent on heavily T2-weighted imaging that selectively displays static or slow-moving fluid-filled structures

Thick-Slab, Single-Shot TSE (or Fast Spin-Echo) T2-weighted Sequence (Projective MRCP)-This technique uses a single, thick (20–50-mm) section that can be obtained in any plane with a single short breath-hold (<3 seconds). This provides an excellent selective display of the whole extrahepatic biliary tract and pancreatic duct with no respiratory artefacts, few susceptibility effects, and a relatively good in-plane resolution.[44]

There are some drawbacks associated with this single-section technique: (*a*) It requires good patient cooperation for breath-holding. (*b*) Ductal visibility may be degraded by possible overlap with other fluid-containing organs (ie, the stomach and duodenum) and the presence of ascites or peripancreatic exudates in the field of view (C) No signal from solid organs is displayed.

Multisection Single-Shot Thin-Slab TSE (or Fast Spin-Echo) T2-weighted Sequence- In this sequence, a series of contiguous thin sections (generally 4 mm thick) is acquired with a shorter echo time and echo train length than with the thick-slab acquisition technique. Therefore, it allows

the depiction of not only the ducts but the solid organs. Image quality is less dependent on patient cooperation and nearly unaffected by specific artefacts (chemical shift, magnetic susceptibility) or motion, the individual sections being acquired during a single shot lasting less than 1 second. However, in cooperative patients, it is preferable to acquire each section during a single breath-hold to reduce misregistration artefacts.[45]

Imaging of Pancreatic Parenchyma

T2-weighted Sequences

Breath-hold or respiratory-triggered multisection, thin-section, HASTE T2-weighted sequences with short (40–80 msec) and long (325 msec) echo times are obtained in the axial and coronal planes. With these sequences, fluid-filled lesions in or around the pancreas are demonstrated and the common bile duct and the pancreatic duct are shown in cross-sections and may be used to guide the acquisition of the MRCP series. Because it may decrease the conspicuity of pancreatic contours, fat suppression is not systematically applied unless peripancreatic exudates are suspected.[46]

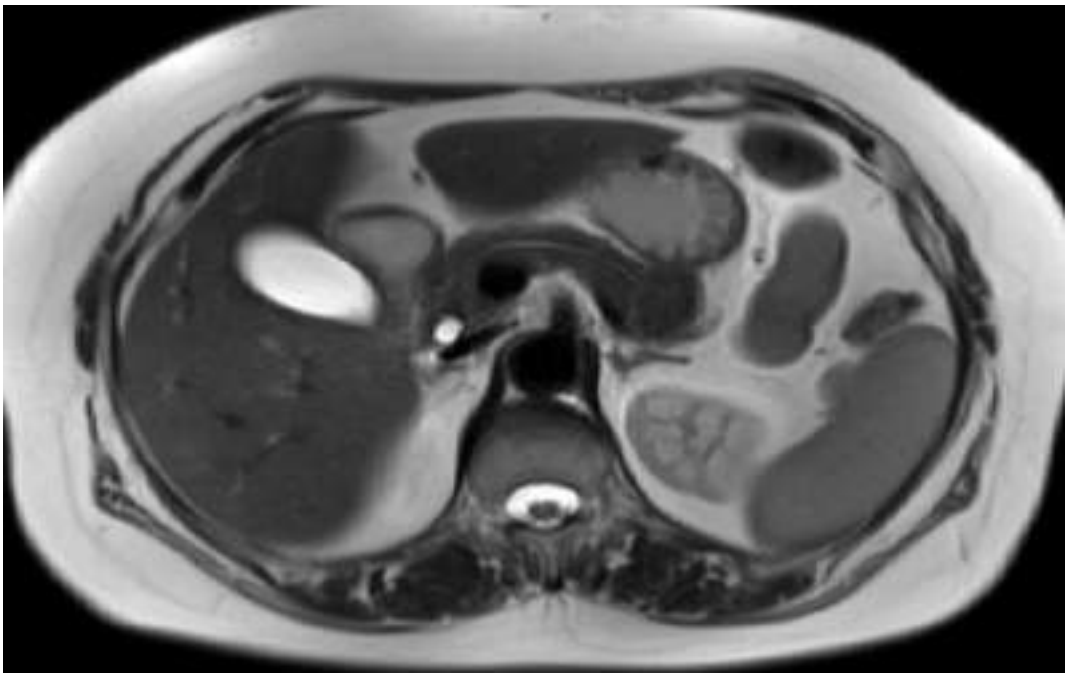


Fig 5: Haste T2 axial image shows normal pancreatic duct (Source – Control number 47 as in the masterchart)

T1-weighted Sequences

These images are obtained either with a fat-suppression TSE T1-weighted sequence using multiple breath-hold acquisitions or with a single-breath-hold gradient-echo (GRE) sequence with a water-stimulation prepulse. Fat suppression or selective water stimulation (*a*) improves the delineation of pancreatic borders and the pancreas itself, which appears homogeneously bright compared with surrounding low-intensity fat ; (*b*) is excellent for identifying pancreatic masses or focal pancreatitis (less intense than normal high-intensity pancreas), and (*c*) is suitable for contrast-enhanced studies. However, breath-holding requires patient cooperation, and these sequences are less accurate in demonstrating focal disease in the presence of diffuse chronic pancreatitis because of the homogeneous low signal intensity of the fibrotic pancreas.[47]

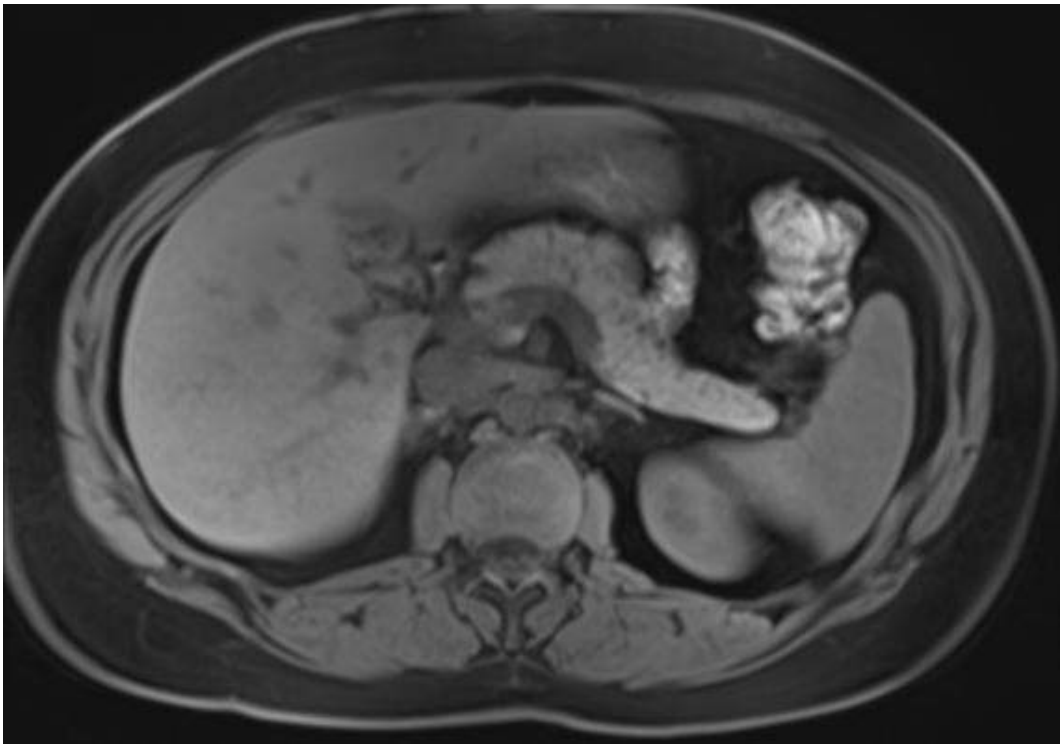


Fig 6: Axial T1-weighted fat-suppressed spoiled gradient-echo image shows normal signal intensity of pancreas is markedly hyperintense to liver and to spleen. (Source –Control number 47 as in the masterchart)

AIMS AND OBJECTIVES

To study the morphological, ductal and signal intensity pattern changes of pancreas in patients with chronic pancreatitis

REVIEW OF LITERATURE

Chronic pancreatitis (CP) involves progressive inflammatory changes in the pancreas characterized by chronic unrelenting pain with episodic flares, impairment of exocrine and endocrine function. The diagnosis of CP relies on relevant symptoms and diagnostic modalities to assess pancreatic structure and function. An important factor for imaging of CP is the presence of fatty infiltration and parenchymal atrophy, which are two commonly observed manifestations of the disease.

A theoretical advantage of MR in the diagnosis of the pancreatic disease is the high contrast resolution between different tissues it makes possible and its ability to show altered signal intensity even in the absence of gross morphological abnormality.[48]

Benoıt P. Gallix et al did a study to evaluate the accuracy of signal intensity(SI) analysis on unenhanced fat-suppressed T1-weighted MR images in the diagnosis of pancreatic disease and to compare subjective interpretation with different quantitative measurements. They concluded that the pancreas–liver ratio is the best quantitative means of distinguishing normal from the abnormal pancreas. Visual observation by experienced observers (qualitative Measurement) was just as accurate as a quantitative measurement. Detection of pancreatic pathology can be made with high accuracy by visually comparing the SI of the pancreas with that of the normal liver.[49]

Anil K. Dasyam et al did a study and concluded that Chronic pancreatitis (CP) remains a diagnostic challenge as clinical symptoms are non-specific, histopathological appearances are varied and pathogenesis remains incompletely understood. Multiple classifications and grading systems have been proposed for CP, but none leverage the full capabilities of cross-sectional imaging modalities and are not widely accepted or validated. CT and MRI/MRCP are useful in identifying a wide spectrum of histopathological changes in CP and can also assess the

exocrine reserve of the pancreas. Advanced MRI techniques such as T1 mapping and extracellular volume fraction can potentially identify early CP. Cross-sectional imaging-based severity scoring can quantify CP disease burden and may have positive implications for clinicians and researchers. In this review, they discuss the need for cross-sectional imaging-based severity scoring for CP, the role of CT, and MRI/MRCP in the assessment of CP and how these modalities can be used to obtain severity scoring for CP. They summarize relevant information from recently published CT and MRI/MRCP reporting standards for CP, and from international guidelines for cross-sectional imaging and severity scoring for CP.[50]

Temel Tirkes, Evan L. Fogel did a study and concluded that there was a significant difference in the signal intensity ratio of the pancreas to the spleen (SIRp/s) between the normal and low bicarbonate groups ($p < 0.0001$). A significant positive correlation was found between pancreatic fluid bicarbonate level and SIRp/s ($p < 0.0001$). SIRp/s of 1.2 yielded sensitivity of 77% and specificity of 83% for detection of pancreatic exocrine dysfunction (AUC: 0.89). T1-weighted MR signal of the pancreas has high sensitivity and specificity for the detection of parenchymal abnormalities related to exocrine dysfunction and can therefore be helpful in the evaluation of suspected early CP.[51]

Hekimoglu K et al did a study to compare the diagnostic potential of one of the new MR sequences in magnetic resonance cholangiopancreatography (MRCP) procedure and endoscopic retrograde cholangiopancreatography (ERCP) with the review of current kinds of literature. They concluded that MRCP is used with increasing frequency as a non-invasive alternative to ERCP and the diagnostic results of MRCP with a heavily T2-weighted TSE MR sequence and ERCP are comparable with high accuracy in various hepatobiliary pathologies.[52]

Soto JA did a study to determine the role of three-dimensional fast spin-echo magnetic resonance (MR) cholangiopancreatography in the

evaluation of the normal and abnormal pancreatic duct. A non-breath-hold MR cholangiopancreatographic technique with the use of a body coil was compared with direct pancreatography performed with endoscopic retrograde cholangiopancreatography in 37 patients. They concluded that MR cholangiopancreatography can accurately demonstrate the normal pancreatic duct as well as various pancreatic duct abnormalities.[53]

Calvo MM et al did a study to prospectively determine the diagnostic accuracy of magnetic resonance cholangiopancreatography (MRCP)in the evaluation of the normal and diseased pancreatic duct.

Patients seen during 6 months with a diagnosis of the biliary tract or pancreatic disease underwent endoscopic retrograde cholangiopancreatography (ERCP) after a previous MRCP. The pancreatic duct was evaluated with both techniques in 37 patients.

They concluded that MRCP is an accurate technique for evaluating the normal or diseased pancreatic duct and for determining the underlying disease.[54]

Lomanto D et al did a study to assess the feasibility of MRCP versus ERCP in the diagnosis of the biliary tract and pancreatic diseases. The MRCP was performed with a 0.5T superconducting magnet. When the neoplastic disease was detected, additional images on axial planes were acquired. MRCP allowed images of diagnostic value to be obtained in all the cases. They concluded that MRCP can be considered a technique able to completely replace diagnostic ERCP.

Further studies are necessary for a better evaluation of the potential advantages and disadvantages of this technique.[55] Temel Tirkes et al did a study to determine if the T1 relaxation time of the pancreas can detect parenchymal changes in mild chronic pancreatitis (CP). T1 maps were obtained in all patients using the same 3D gradient-echo technique on the same 3T scanner. T1 relaxation times, the fat signal fraction (FSF), and anterior-posterior (AP) diameter was correlated with the clinical diagnosis of CP. The T1 the relaxation time of the pancreatic parenchyma was

significantly increased in patients with mild CP. Therefore, T1 mapping might be used as a practical quantitative imaging technique for the evaluation of suspected mild CP.[56]

Sivasubramanian Srinivasan et al did a study in Recent Advances in Pancreatic MR Imaging: A Guide on How, When, and Why to Perform. They concluded that MRI has a problem-solving role in pancreatic imaging in routine clinical practice. Advanced diffusion techniques can localize and characterize focal and diffuse pathologies serving as a useful adjunct to conventional MRI. Secretin MRCP can improve visualization of ductal anatomy and pathologies and can potentially demonstrate pancreatic leak. Advanced imaging sequences and specialized sequences are useful in the quantitative and semiquantitative assessment of pancreatic fat, iron, fibrosis, and tissue characters that can help in better characterization of diffuse pancreatic diseases shortly early changes of acute pancreatitis.[57]

Sriluxayini Manikkavasakar et al did a study in which they emphasized the role of MRI in imaging all types of acute and chronic pancreatitis, pancreatitis complications, and other important differential diagnoses that mimic pancreatitis. MRI is a valuable alternative modality, with at least equal diagnostic performance to CT for the diagnosis and follow-up of acute and chronic pancreatitis. The recent development of new respiratory gating techniques, motion resistant pulse sequences, and additive advantages of MRCP imaging protocols make MRI a very accurate investigation modality for assessing patients with pancreatitis; particularly acutely ill patients unable to breathe-hold. MRI is a non-ionizing cross-sectional imaging method with a safer intravenous contrast profile. This is particularly important in patients with acute pancreatitis, who often have a concomitant renal impairment of some degree and often require repeated follow-up imaging. Additionally, MRI offers higher sensitivity for the diagnosis of subtle peripancreatic oedema) and early manifestations of chronic pancreatitis.[58]

TEMEL TIRKES, M.D. did a study and concluded that the diagnosis of CP can be challenging since biopsy of the pancreas is not performed, and laboratory studies and imaging procedures may be normal during the early stage of the disease. The diagnosis of the advanced CP is confirmed if there are calcifications within the pancreas on plain abdominal films or computed tomography (CT) scan, an abnormal pancreatogram or an abnormal secretin pancreatic function test in subtle cases of early pancreatitis. Identification of early-stage CP and its treatment may delay or prevent morbidity secondary to CP. In settings where MRI/MRCP is available and of high quality, it may be the imaging test of choice, allowing for assessment of ductal changes and potentially obviating the need for an invasive procedure. Stimulation of the pancreas using intravenous secretin may improve the diagnostic accuracy in the detection of ductal and parenchymal abnormalities seen in CP. T1 signal intensity changes in the pancreatic parenchyma may precede ductal abnormalities and may detect early CP. Ultrasound and CT are best for the late findings of CP but are limited in the diagnosis of early or mild pancreatitis. A contrast-enhanced CT scan can rule out other causes of pain that mimic CP and is helpful for the diagnosis and complications of CP. There is a need for a EUS-like MRI staging system for CP, combining the ductal findings with the parenchymal changes.[59]

Samer Alkaade et al did a study that normal pancreatic function testing cannot exclude abnormal MRI/MRCP, especially focal findings of chronic pancreatitis. Further studies are needed to verify the significance of these findings and establish MRI/MRCP imaging criteria for the diagnosis of chronic pancreatitis.[60]

Temel Tirkes Chen Lin et al did a study that showed that ECV fraction and tissue relaxometry information provided by T1 mapping can provide quantitative metrics for determining the presence and severity of acinar cell loss and fibrosis and aid in the diagnosis of CP. Including these quantitative imaging techniques as part of the routine MRCP evaluation

can help radiologists by increasing sensitivity and specificity in the evaluation of CP. Our study is the first to our knowledge to explore the potential benefit of these new imaging techniques. Prospective studies should be performed to validate these results using histopathologic correlation, different T1 mapping sequences, and MR scanners from different vendors.[61]

Rohan Kamat, Pankaj Gupta et al did a study to determine sensitivities of the diagnostic tests for CP in descending order are ERCP (82%), EUS (81%), MRCP (78%), MDCT (75%), and ultrasound (67%). Specificities for the diagnosis of CP are MRCP (96%), ERCP (94%), MDCT (91%), and EUS (90%). MRI, MDCT, EUS, and ERCP were found to have high diagnostic accuracy. Among them, the latter two had the highest scores. However, due to the invasive nature of these tests, MDCT and MRCP are preferred over these tests. MDCT and MRCP are the first-line investigations in CP. ERCP is generally used only when therapeutic interventions are planned. ERCP for the diagnostic purpose has been substituted by EUS, MDCT, and MRCP.[62]

Axon AT, Classen M et al did a study and concluded that Main duct abnormalities include dilation, narrowing, strictures, filling defects and leaks or cavities. Identification of these abnormalities can be subjective as their definitions are not clear. Average normal main duct diameters were 3.6, 2.7 and 1.6 mm in the head, body and tail respectively, though the upper limited normal may be as high as 6.5, 5 and 3 mm in the head, body and tail, respectively. Main duct dilation is either general (involving more than 2/3 of the main duct) or local (< 2/3 of the ducts). Severe dilation was defined as being > 1 cm.[63]

Monica Cheng, Mark A Gromski et al studied that There was no significant difference between the T1 relaxation times of Cambridge 0 and 1 group (p = 0.58). There was a significant difference (p = 0.0003) in the mean T1 relaxation times of the pancreas between the combined Cambridge 0 and 1 (mean = 639 msec, 95% CI: 617, 660) and Cambridge 2 groups (mean

= 726 msec, 95% CI: 692, 759). There was significant difference (p = 0.0009) in the mean T1 relaxation times of the pancreas between the Cambridge 0 (mean = 636 msec, 95% CI: 606, 666) and Cambridge 2 groups (mean = 726 msec, 95% CI: 692,759) as well as between Cambridge 1 (mean = 643 msec, 95% CI: 608, 679) and Cambridge 2 groups (mean = 726 msec, 95% CI: 692,759) (p = 0.0017). Bland–Altman analysis showed measurements of one reader to be marginally higher than the other by 15.7 msec (2.4%, p = 0.04). T1 mapping is a practical method capable of quantitatively reflecting morphologic changes even in the early stages of chronic pancreatitis, and demonstrates promise for future implementation in routine clinical imaging protocols.[64]

In a previous study by Edge MD, ductal dilatation was graded as normal, mild, moderate, or severe. However, like atrophy, grading ducts dilation is also challenging given the lack of widely accepted value for normal and given expected age-related changes. 3 or 3.5 mm has been considered as the threshold by some authors in the past. Irrespective of the diameter (i.e., even if less than 3 mm), an increase in calibre upstream to a stricture or intraductal calculus must be considered dilated.[65]

MATERIALS AND METHODS

SOURCE OF DATA –

All patients who are diagnosed or suspected to have chronic pancreatitis and are referred for MRI abdomen to the Department of Radiodiagnosis, DMCH Ludhiana for a period of one and half year from 1st Dec 2019 to 31st May 2021.

METHOD OF COLLECTION OF DATA

The clinically or biochemically diagnosed patients were assessed for morphological changes in the pancreas with emphasis on signal intensity pattern and ductal changes in chronic pancreatitis. The signal intensity pattern of the patients with chronic pancreatitis was compared with age and sex-matched controls to assess and define the range of values in patients and the control population.

Inclusion criteria

1) Clinically diagnosed patients of chronic pancreatitis.

2) Patients with biochemical, alternative imaging and other clinical parameters suggestive of chronic pancreatitis.

Exclusion criteria:

1) If the patient refuses to participate in the study

2) Contraindications to MRI as per the checklist on the departmental consent form

3) Acute pancreatitis

4) Known cystic or solid pancreatic neoplasm

5) Incomplete or suboptimal quality of MRI examinations

MRI PROTOCOL

All patients underwent MR imaging using the following protocol: The patients were subjected to a multiplanar, multi sequential scan using a SIEMENS MAGNETOM SKYRA 48 CHANNEL 3 Tesla MRI.

	T1 vibe Fat sat	T1 vibe In –out phase	T2 haste fat sat	T2 haste traction	T2 haste coronal	T2 haste thick slab	Diffusion
TR	3.31 ms	4.34 ms	2000ms	2000ms	2000ms	4500ms	5700ms
TE	1.3 ms	1.34,2.76 mm	91ms	91ms	91ms	734ms	48ms
FOV (R)	380 mm	380 mm	350 mm	360 mm	350 mm	260 mm	380 mm
FOV (P)	81.3%	81.3%	78.1%	78.1%	100%	100%	80.6%
SLICE	72	72	30	30	35	1	30
SLICE THICK	3mm	3mm	5mm	5mm	5mm	50mm	5mm
GAP	1.1 mm	1.1mm	2.5mm	2.5mm	0.5mm	2.5mm	2.5mm
FLIP ANGLE	9^0	9^0	160^0	160^0	160^0	180^0	
FAT SAT	SPAIR	NONE	SPAIR	NONE	NONE	FATSAT	SPAIR
MATRIX	182*320	182*320	200*320	200*320	288*320	314*448	108*134
FLOW COMP	NO	NO	NO	NO	NO	NO	
PHASE ENC	AP	AP	AP	AP	R-L	R-L	AP
ORIENTATION	Transverse	Transverse	transverse	transverse	coronal		Transverse

MRI Technique

All MR examinations were performed with 3 T superconducting magnets using a torso multi-coil array. Breath-hold unenhanced fat-suppressed GRE T1 weighted images were obtained in an axial plane using a half field of view ranging from 32 to 48 cm. GRE breath-hold images were obtained with a TR of 150- 270 msec, a TE of 4.2 msec and a flip angle of 700. The matrix size was 256*192, one excitation was acquired, section thickness was 7 mm with no intersection gap for an acquisition time ranging from 19 to 26 seconds. Flow compensation, superior and inferior spatial saturation pulses and fat suppression were used.

Patient Selection

Fast spoiled GRE fat-suppressed T1 weighted images of the pancreas were taken in 300 consecutive patients referred for MR examination of the upper abdomen for 1.5 years. The data from 210 patients were excluded from the analysis. The reasons for this included pancreatic malignancy, acute pancreatitis, absence of definitive diagnosis, incomplete MR examinations, and images unacceptable in their quality. The final study population consisted of 90 patients (53 men and 37 women; age range, 20-50 years; mean age, 32.3 years). Based on clinical, biological, imaging (other than MRI), and pathologic criteria 45 of them were considered to have a clinically abnormal pancreas and 45 had no evidence of pancreatic disease. There were 8 women and 37 men with a mean age of 40.29 years (range, 24–52 years) in the normal group. There were 7 women and 38 men with a mean age of 30.33 years (range, 22–56 years) in the pathological group. The prospective study was conducted on 45 patients diagnosed with chronic pancreatitis on basis of clinical and biochemical parameters. MRCP was performed to assess the signal, morphological and ductal changes. 45 matched controls were also assessed for signal pattern of the pancreas to derive cut off values in patients with chronic pancreatitis.

Out of 45 cases, 25 patients were diagnosed clinically based on chronic epigastric pain, 15 patients were diagnosed biochemically, 5 patients were diagnosed on previous imaging such as ultrasound and CT.

45 controls were taken those who underwent MRCP for other indications including pain abdomen, tumour assessment, other pathologies with no clinical or biochemical evidence to suggest chronic pancreatitis.

Signal changes, as well as ductal changes, were assessed qualitatively as well as quantitatively in both cases and controls, further results were documented to find out better parameters in early diagnosis of chronic pancreatitis.

Qualitative Analysis

The examinations were all reviewed on a workstation multiplanar, multisequential scan using a SIEMENS MAGNETOM SKYRA 48 CHANNEL 3 Tesla MRI.

Visual and subjective assessment of the pancreatic signal was done in comparison to the liver, spleen, and paraspinal muscles in cases and controls. The patients with liver disease were excluded. The pancreatic signal was graded as more than liver, equal to the liver, mildly less than liver, Significantly less than the liver (Similar to muscles). Signal more or equal to the liver was considered normal in both cases and controls. Relative lowering of the signal was considered pathological.

Ductal morphology: The main pancreatic duct was assessed for calibre, contour and stricturing.

The presence or absence of side duct branches was also assessed and documented.

1. Ductal calibre:

1) (<3.5 mm) -Normal

2) 3.5 to 7 mm- Mild

3) >7 mm – Severe

2. No of visualized/dilated pancreatic duct side branches-

< 3 side duct branches: Normal.

> 3 side duct branches: Abnormal.

3) Pancreatic duct stricture:

Focal narrowing of the main pancreatic duct on multiple planes with or without upstream dilatation was defined as a stricture.

The number of strictures and the most significant strictures was documented.

4. Pancreatic duct contour- PD Pancreatic duct contour was graded as

Regular: Smooth contour in the entire visualized length

Irregular: Undulating / tortuous contour

5. Location of Pancreatic duct strictures-

a) None

b) Head, Neck, body or tail.

6) Intraductal calculus – A measurable signal void in the duct was defined as an intraductal calculus. Intraductal calculus is suspected when there is a measurable loss of signal void in the course of MPD in the head, neck, body or tail region.

7. Pancreatic thickness- The measurement was performed in the region of the pancreatic body to the left margin of the vertebral body.

The severity of the parenchymal atrophy was graded as :

1. </= 7mm : Severe
2. 7-14 mm: Moderate
3. 14-21 mm: Mild
4. 21 mm: Normal.

8. Pancreatic calcifications: Punctate foci of signal void in the parenchyma were defined as pancreatic calcifications.

The severity of the ductal changes was classified in our study as per the Cambridge classification for chronic pancreatitis. The Cambridge classification (Table 1), which was developed for endoscopic retrograde cholangiopancreatography, has been suggested for translational use in CT and MRI or MR cholangiopancreatography interpretation by recent guidelines of the American Pancreatic Association.

Table 1: Cambridge Classification

Cambridge classification	Main PD	Abnormal side branches
Grade 0,normal	Normal	None
Grade 1,equivocal	Normal	Fewer than three
Grade 2,mild CP	Normal	Three or more
Grade 3, moderate CP	Abnormal	More than three
Grade 4, Severe CP	Abnormal	One or more large cavity, obstruction, filling defects, severe dilatation or irregularity

CP- chronic pancreatitis, PD – Pancreatic duct

Quantitative Image Analysis

Standard regions of interest (ROIs) were made with an area of 25–50 mm2, taking care to avoid the inclusion of vessels in the liver on the one hand or fat located between pancreas lobulations on the other. Three SI measurements were obtained in the head, body, and tail of the pancreas and averaged. When an area of focal hypointensity was detected in the pancreas, an additional measurement was obtained from this area. The lower of the two results were used to calculate the different ratios. An average of three measurements was also obtained from the liver, spleen and paraspinal muscle used as comparison tissue. If possible, the ROI was performed in the liver, spleen and paraspinal muscles on the same section as in the pancreas. Otherwise, the SI of the liver or spleen was measured on the closest section available. The pancreas–liver, pancreas–spleen, pancreas –paraspinal muscle SI ratios were calculated as follows: pancreas to comparison tissue SI ratio [(SI pancreas/SI comparison tissue) – 1]. Using this formula, the ratio is equal to 0 when the pancreas has the same SI as the comparison tissue. Different ratios were calculated and mean values were obtained. Using a threshold value of 0, if the signal intensity ratio of the pancreas to the liver was equal to or more than 0 then

it was considered normal, if the signal intensity ratio of the pancreas to the liver was less than 0 then it was considered abnormal.

STATISTICAL ANALYSIS

All statistical calculations were done using SPSS 21 (Statistical Package for the Social Science) version statistical program for Microsoft Windows. Data were described in terms of range; mean ±standard deviation (± SD), frequencies (number of cases) and relative frequencies (percentages) as appropriate. Comparison of quantitative variables between the study groups was done using the Student t-test. For comparing categorical data, Chi-square ($\chi 2$) test was performed and an exact test was used when the expected frequency is less than 5. A probability value (p-value) less than 0.05 was considered statistically significant.

OBSERVATIONS AND RESULTS

1) Demographic Data

There was no significant correlation between the age of the patient, their sex, and the SI of the pancreas in the normal group. There were 8 women and 37 men with a mean age of 35.53 years ± 8.56 SD in the normal group. There were 7 women and 38 men with a mean age of 32.33year ± 12.41 in the pathological group

Table 2: Mean age value among cases and controls

	Control		Cases		T	p-value
	Mean	SD	Mean	SD		
Age	35.53	8.56	32.33	12.41	-1.424	0.158

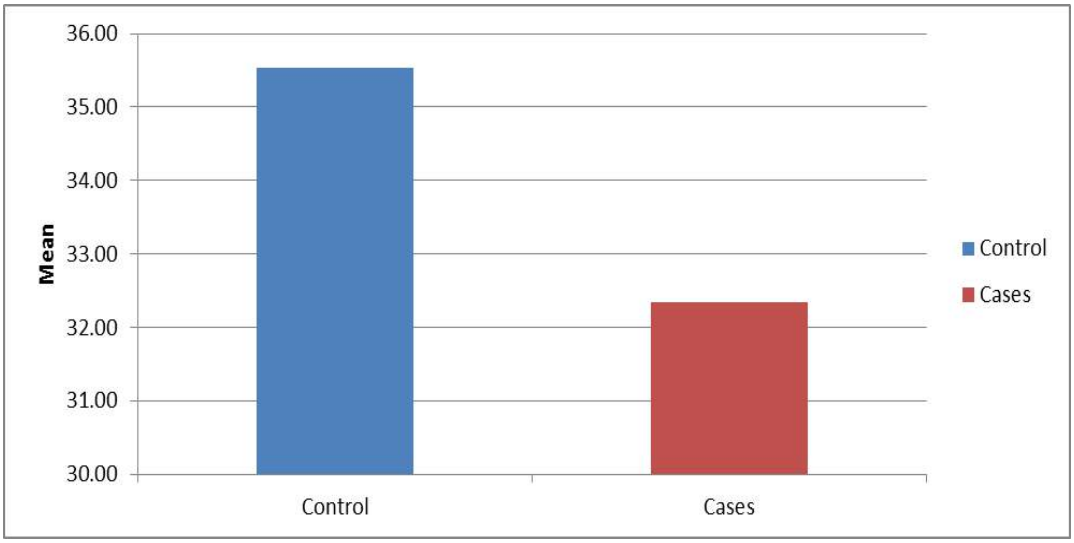


Fig 7: Mean age value among cases and controls

Table 3: Age distribution among cases and controls

		Group				Total	Chi-square value	p-value
		Control		Cases				
Age group	< 30	9	20%	14	31%	35	2.299	0.317
	31-40	19	42%	19	42%	34		
	> 40	17	38%	12	27%	21		
Total		45	100%	45	100%	90		

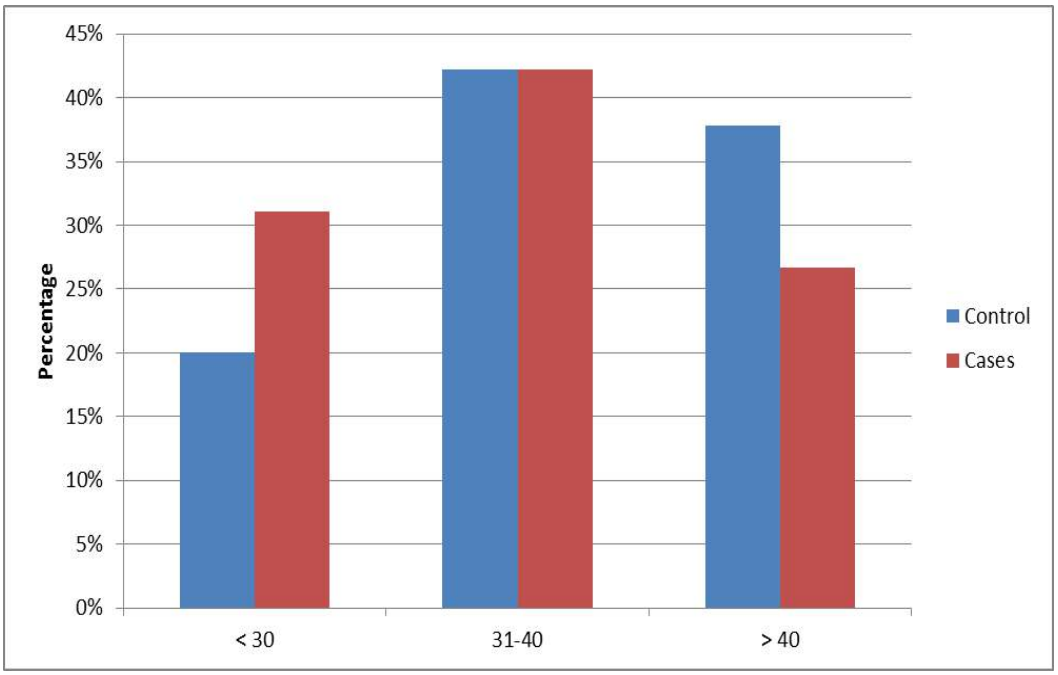


Fig 8: Age distribution among cases and controls

Table 4: Gender distribution among cases and controls

		Group				Total	Chi-square value	p-value
		Control		Cases				
Sex	F	8	18%	7	16%	15	0.08	0.777
	M	37	82%	38	84%	75		
Total		45	100%	45	100%	90		

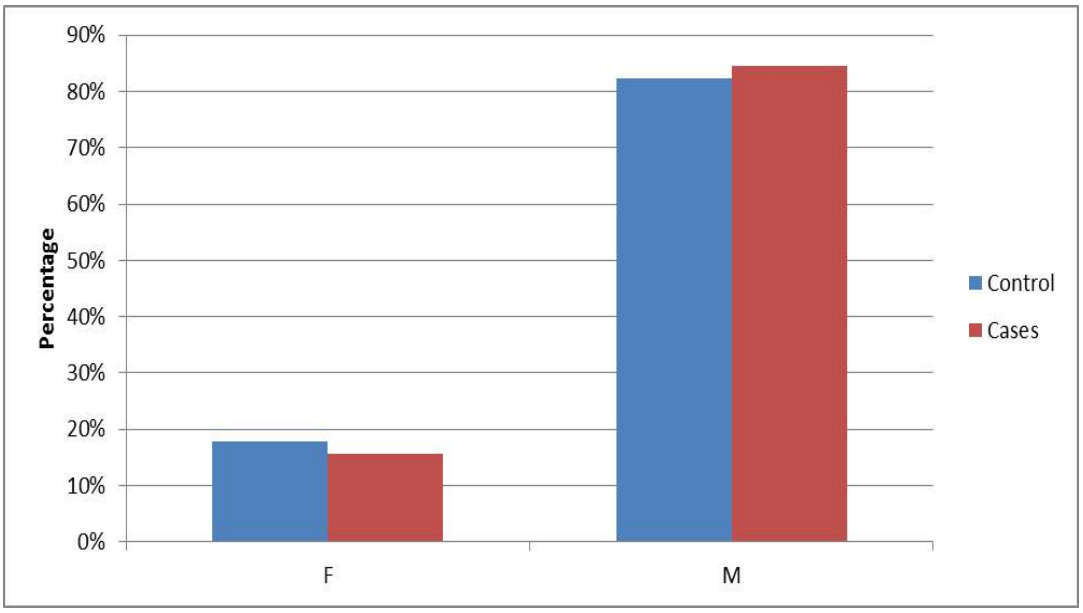


Fig 9: Gender distribution among cases and controls

2) Clinical Data

Out of total 45 cases, 40 % patients were presented with history of weight loss, 38 % patients were presented with complaints of epigastric pain and 22% were presented with steatorrhea.

Table 5: Distribution of patients according to Clinical history

		Cases	
Clinical features	Epigastric pain	17	38%
	Steatorrhea	10	22%
	Weight loss	18	40%
Total		45	100%

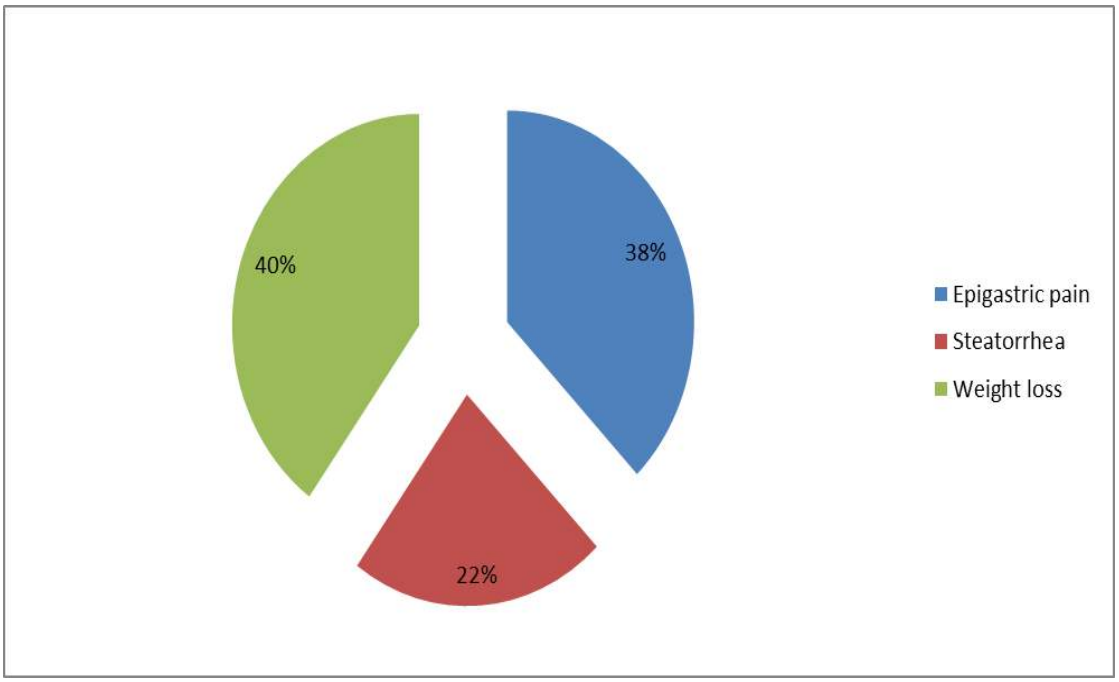


Fig 10 : Distribution of patients according to Clinical history

3) Biochemical profile

Biochemically , elastase levels were low (<200) in 39 cases with mean value of 154.95 ± 20.05 SD. Mean serum amylase and lipase levels were 34.24 ± 6.29 SD and 57.73 ± 12.82 SD in 45 cases respectively. CRP levels were high in both cases as well as controls with mean value of 9.42 ± 6.30 .

Table 6 : Biochemical parameters among cases and controls

	N	Minimum	Maximum	Mean	Std. Deviation
Elastase (mcg/g)	39	117.00	187.00	154.95	20.05
S.Amylase	45	25.00	49.00	34.24	6.29
S.Lipase	45	36.00	95.00	57.73	12.82
CRP	90	1.00	21.00	9.42	6.30

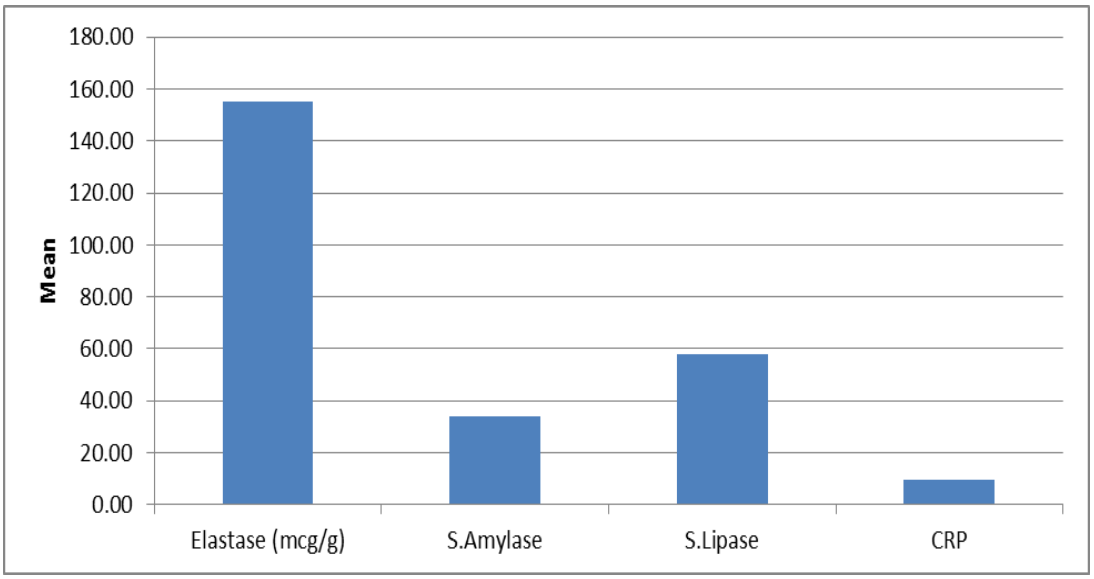


Fig 11: Biochemical parameters among cases and controls

4) Parenchymal Thickness

In our study, the mean pancreatic parenchymal thickness was 13.58 mm ± 5.46 SD in cases and was 22.40 mm ± 1.21 SD in controls respectively.

Table 7: Mean parenchymal thickness among cases and controls

	Control		Cases		t	p-value
	Mean	SD	Mean	SD		
Parenchymal Thickness	22.40	1.21	13.58	5.46	-7.086	0.001

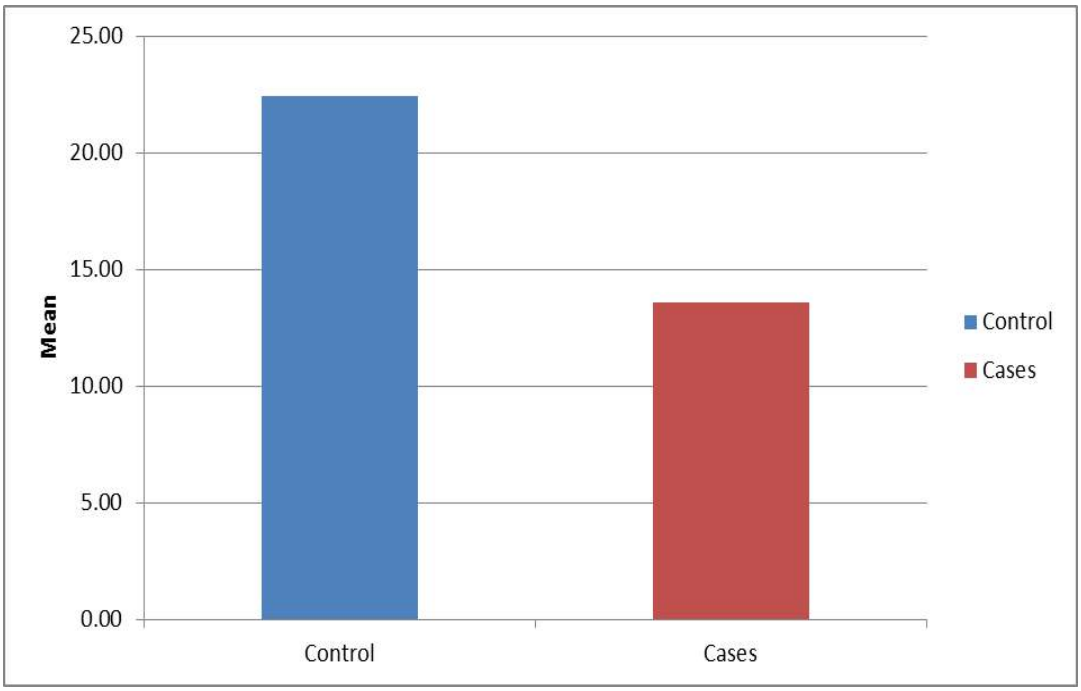


Fig 12: Mean parenchymal thickness among cases and controls

5) T1 Signal intensity

The mean value of SI of pancreas in 45 cases was 201.51 ± 93.78 SD with significant p value. The mean value of SI of pancreas in 45 controls was 347.09 ± 43.71 SD with significant p value.

Table 8: Pancreatic parenchymal T1 SI

	Control		Cases		Z	p-value
	Mean	SD	Mean	SD		
Parenchymal T1 SI(P)	347.09	43.71	201.51	93.78	-9.438	0.000
T1 SI Liver	289.53	42.59	242.91	96.54	-2.964	0.004
T1 SI Spleen	261.73	33.69	213.49	88.19	-3.428	0.001
T1 SI PSM	219.82	14.09	195.40	76.53	-2.105	0.038

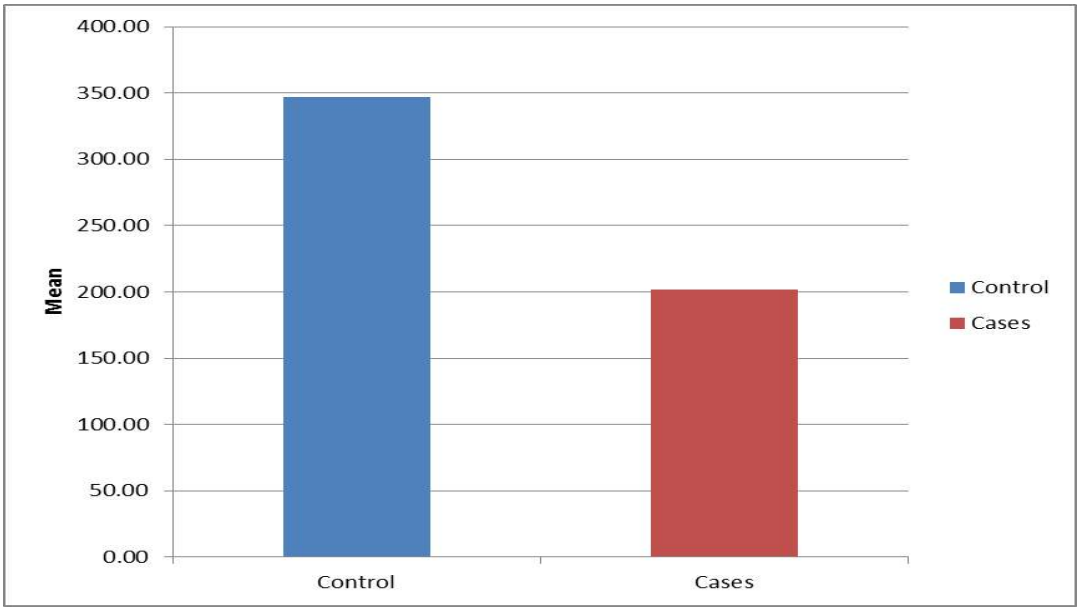


Fig : 13 T1 signal intensities of pancreas, liver, spleen and paraspinal muscles

The pancreas–liver, pancreas–spleen, pancreas –paraspinal muscles SI ratios were calculated as follows: pancreas to comparison tissue SI ratio [(SI pancreas/SI comparison tissue) – 1]. Using this formula, different ratios were calculated and mean values were obtained.

Table: 9 Signal intensity ratios of pancreas to liver, spleen and paraspinal muscles

	Control		Cases		Z	p-value
	Mean	SD	Mean	SD		
SIR(P/L)-1	0.21	0.13	-0.18	0.19	-11.282	0.000
SIR(P/S)-1	0.33	0.11	-0.05	0.21	-10.777	0.000
SIR(P/PSM)-1	0.58	0.22	0.02	0.21	-12.516	0.000

The mean values between cases and controls of pancreas–liver ratios were -0.18 ± 0.19 SD vs. 0.21 ± 0.13, respectively with significant p value.

The mean values between cases and controls of Pancreas–spleen ratios were -0.05 ± 021 SD vs. 0.33 ± 0.11, respectively with significant p value.

The mean values between cases and controls of Pancreas to paraspinal muscles ratios were 0.02 ± 0.21 SD vs. 0.58 ± 0.22 SD respectively, with significant p value.

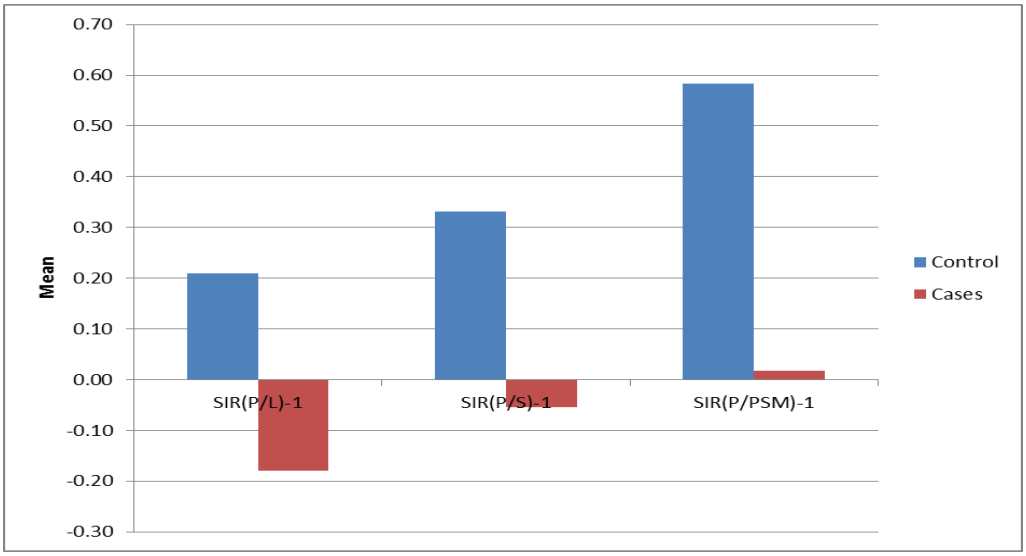


Fig: 14 Signal intensity ratios of pancreas to liver, spleen and paraspinal muscles

6) GRAPH SHOWS ROC CURVES FOR QUANTITAIVE ASSESSMENT OF PANCREAS USING LIVER AND SPLEEN AS COMPARISON TISSUE

The pancreas–liver SI ratio was significantly better than the pancreas–spleen ratio throughout the disease group (area under the receiver operating characteristic (ROC) curve SD; 0.87 for pancreas–liver vs. 0.85 for pancreas–spleen, P 0.000).

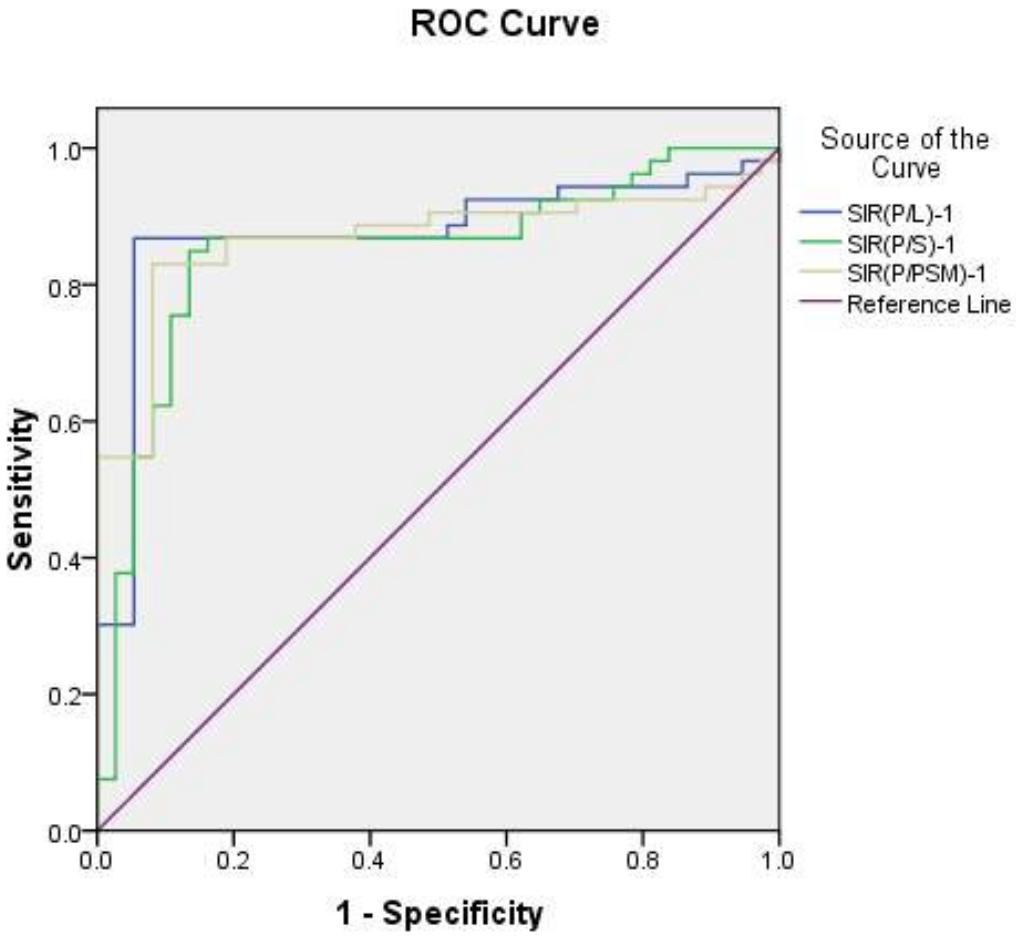


Fig: 15 Graph shows ROC curves for quantitative assessment of the pancreas using liver as comparison tissue in the entire study population (Blue – SIR(P/L)-1, Green- SIR(P/S)-1, Yellow- SIR (P/PSM)-1,Purple- Reference line)

Table- 10 shows Area Under Curves for quantitative assessment of the pancreas using liver as comparison tissue in the entire study population(i.e. SIR(P/L)-1, SIR(P/S)-1, SIR (P/PSM)-1)

Test Result Variable(s)	Area	Std. Error[a]	p-value	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
SIR(P/L)-1	0.874	0.041	0.000	0.793	0.954
SIR(P/S)-1	0.851	0.043	0.000	0.765	0.936
SIR(P/PSM)-1	0.868	0.040	0.000	0.789	0.948

7) Ductal morphology among cases and controls

Main pancreatic duct was maintained in all the cases with 100% sensitivity. However MPD was irregular in contour in 73% of cases, tortous in 4% cases and was regular in 22% of cases with statistically significant P value with chi-square value of 57.273.

Table 11: Pancreatic duct contour among cases and controls

		Group				Total	Chi-square value	p-value
		Control		Cases				
PD Contour	Irregular	0	0%	35	78%	35	57.273	0.0001
	Regular	45	100%	10	22%	55		
Total		45	100%	45	100%	90		

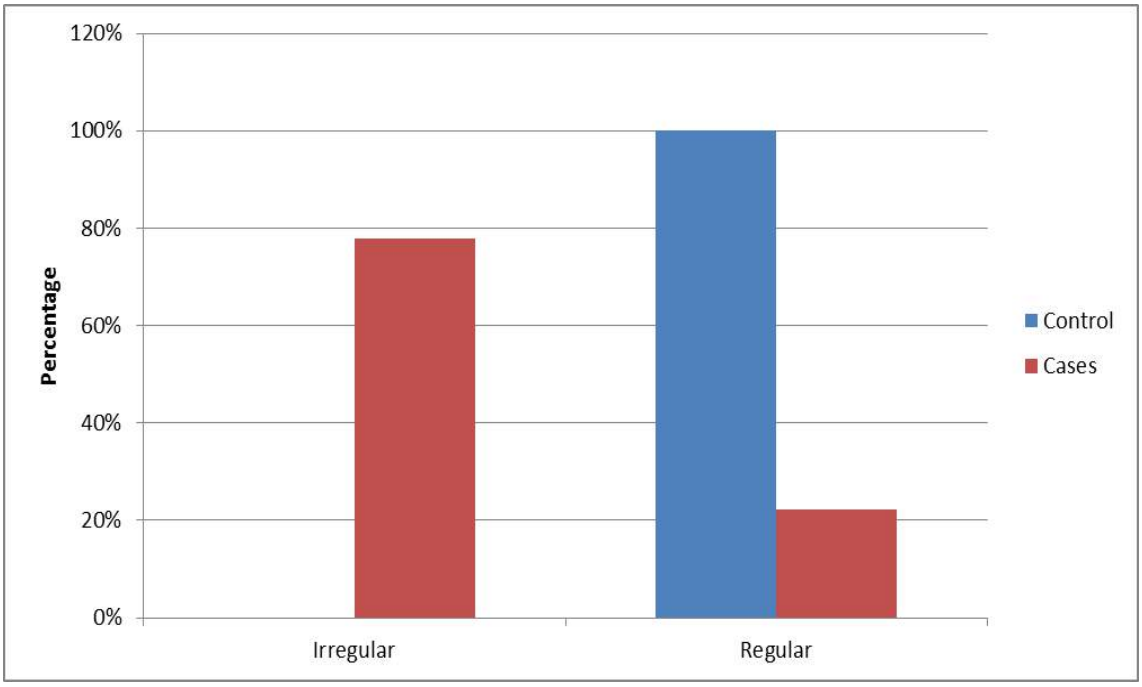


Fig: 16 Pancreatic duct contour among cases and controls

Pancreatic duct morphology was maintained in all the controls with 100% sensitivity. However, PD morphology was altered in 45 cases , out of which multiple side branches were present in 24% of cases, >3 side branches were present in 42% of cases, no dilated side branches were there in 29% of cases , <3 side branches were present in 4% cases with statistically significant p value with chi-square value of 49.655.

Table 12: Pancreatic ductal side branches among cases and controls

		Group				Total	Chi-square value	p-value
		Control		Cases				
No; of PD side branches	> 3	0	0%	19	42%	19	49.655	0.0001
	< 3	0	0%	2	4%	2		
	absent	45	100%	13	29%	58		
	multiple	0	0%	11	24%	11		
Total		45	100%	45	100%	90		

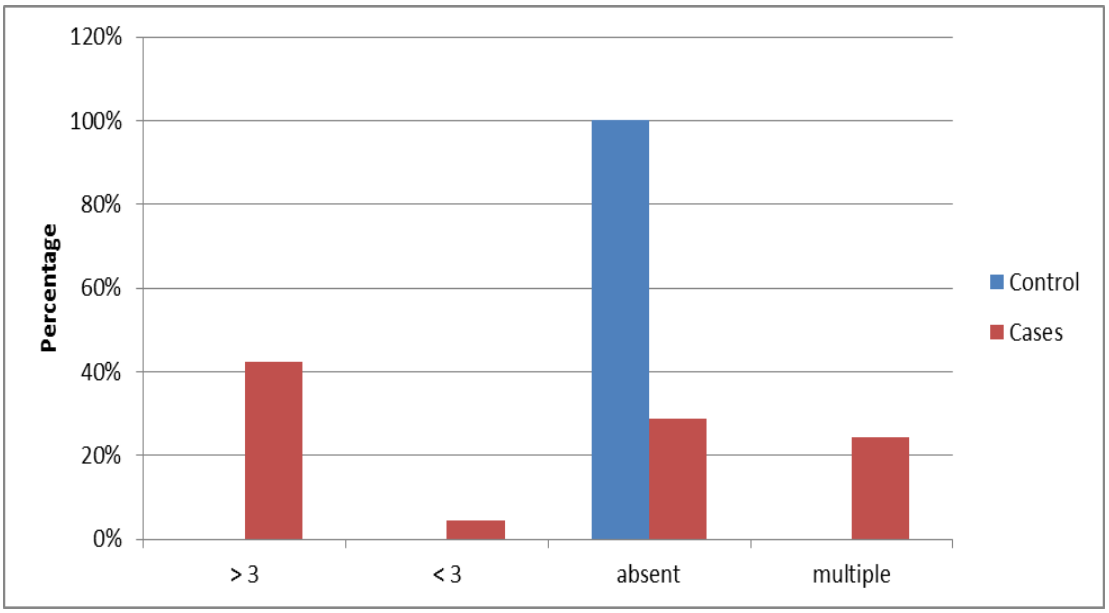


Fig: 17 Pancreatic ductal side branches among cases and controls

Severity of ductal changes was demonstrated as per Cambridge classification system. Grade 4 (Severe) ductal changes were present in 62% of cases. Grade 3 (moderate) ductal changes were present in 22% of cases. Grade 0 (normal) was present in 16% of cases with statistically significant p value with chi-square value of 65.769. Ductal changes were absent in 100% of controls.

Table:13 Severity of ductal changes among cases and controls

		Group				Total	Chi-square value	p-value
		Control		Cases				
Severity of ductal changes	Grade 0 - normal	45	100%	7	16%	52	65.769	0.0001
	Grade 3-Moderate	0	0%	10	22%	10		
	Grade 4-Severe	0	0%	28	62%	28		
Total		45	100%	45	100%	90		

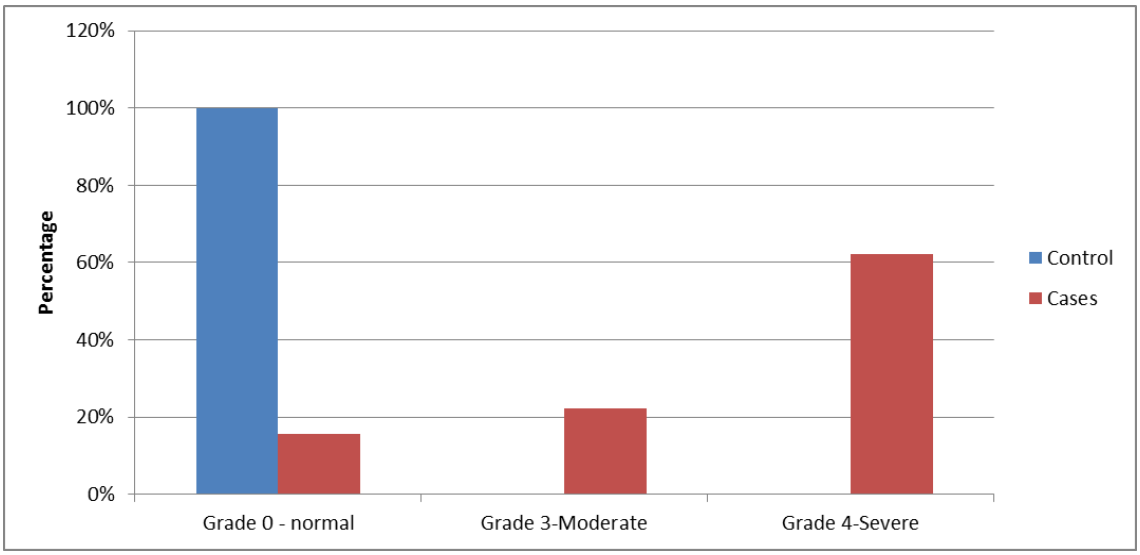


Fig :18 Severity of ductal changes among cases and controls

Pancreatic duct stricture was present in 56% of cases and was absent in 44% of cases with statistically significant p value with chi-square value of 34.615. Pancreatic duct stricture was absent in 100 % of controls.

Table: 14 Intraductal stricture among cases and controls

		Group				Total	Chi-square value	p-value
		Control		Cases				
Intraductal stricture	Absent	45	100%	20	44%	65	34.615	0.0001
	Present	0	0%	25	56%	25		
Total		45	100%	45	100%	90		

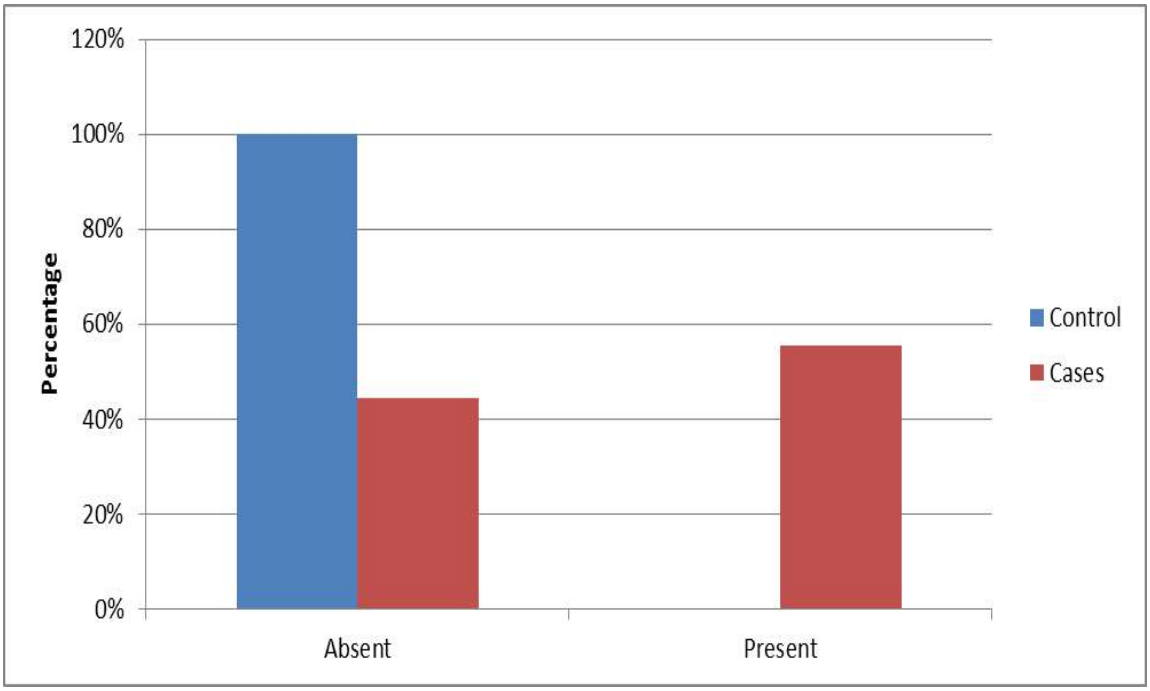


Fig:19 Intraductal stricture among cases and controls

Intraductal Calcification was documented in 20% of cases and was absent in 80 % of cases with chi-square value of 10.000. Intraductal calcification was absent in 100% of controls.

Table: 15 Intraductal calculus among cases and controls

		Group				Total	Chi-square value	p-value
		Control		Cases				
Intraductal Calculus	Absent	45	100%	36	80%	81	10.000	0.002
	Present	0	0%	9	20%	9		
Total		45	100%	45	100%	90		

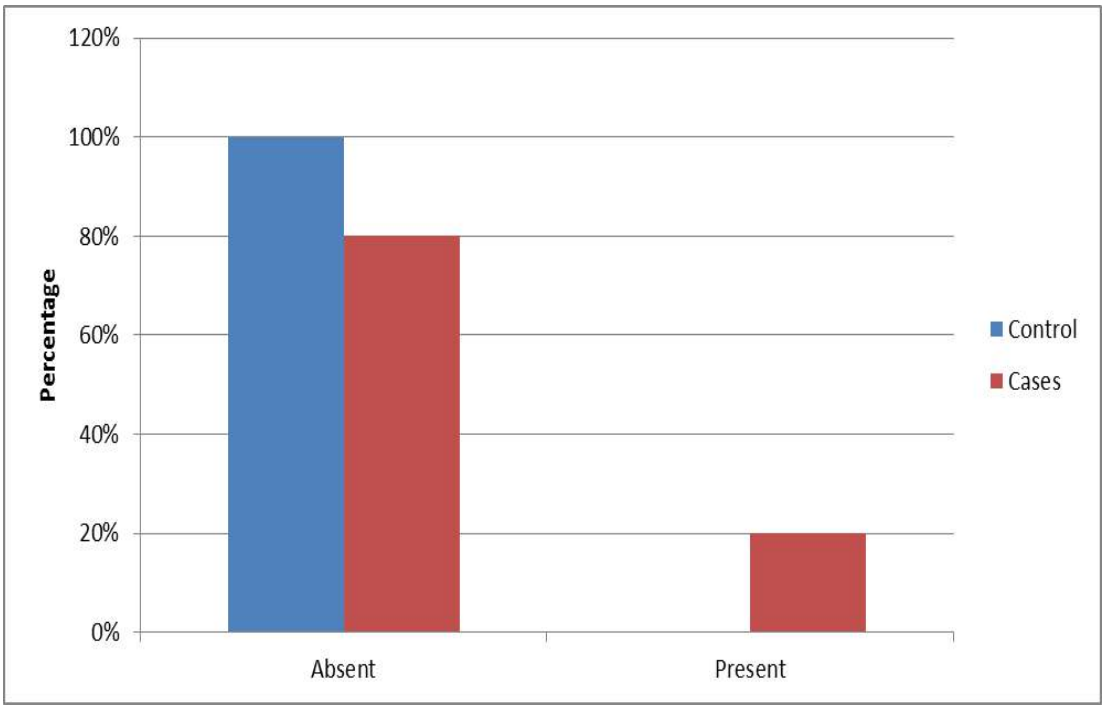


Fig: 20 Intraductal calculus among cases and controls

Lowered T1 signal intensity (SI) was observed in patients with Chronic Pancreatitis on qualitative as well as quantitative analysis. Out of 45 cases, 13% of cases show similar signal intensity as compared to comparison tissues. 0ut of 45 controls, 16% patients show lower signal intensity of pancreas in comparison to other organs. The mean values between cases and controls of pancreas–liver ratios of -0.18± 0.19 SD vs. 0.21 ± 0.13 SD, respectively, and pancreas–spleen ratios of -0.05 ± 0.21 vs. 0.33 ± 0.11 SD, Pancreas to paraspinal muscles ratios of 0.02 ± 0.21 vs. 0.58 ± 0.22 respectively, were significantly different (p 0.001). The pancreas–liver SI ratio was significantly better than the pancreas–spleen ratio throughout the disease group (area under the receiver operating characteristic (ROC) curve SD; 0.87 for pancreas–liver vs. 0.85 for pancreas–spleen, P 0.000).

Table: 16 Only T1 signal changes among cases and controls

		Group				Total	Chi-square value	p-value
		Control		Cases				
Only signal changes	No	0	0%	37	82%	37	75.067	0.0001
	Normal	38	84%	0	0%	38		
	Yes	7	16%	8	18%	15		
Total		45	100%	45	100%	90		

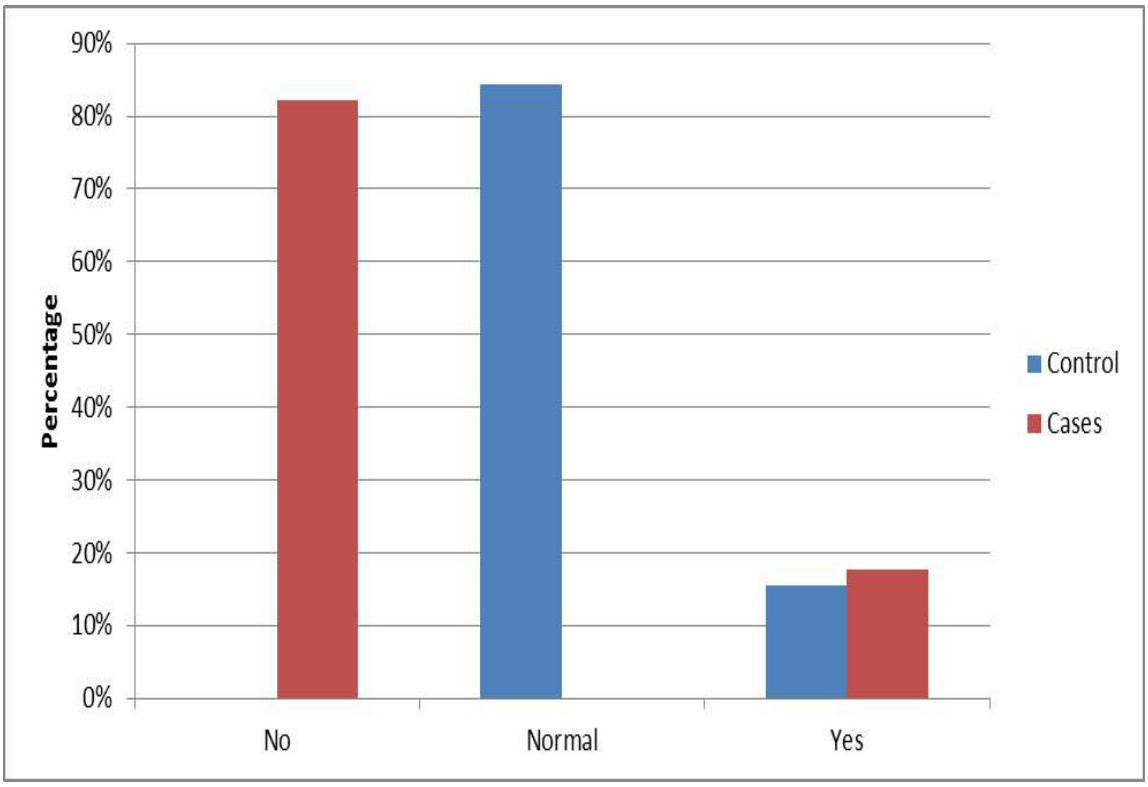


Fig: 21 Only T1 signal changes among cases and controls

Table: 17 Both signal as well as ductal changes among cases and controls

		Group				Total	Chi-square value	p-value
		Control		Cases				
SI + ductal changes	Absent	38	84%	0	0%	38	75.067	0.0001
	Signal	7	16%	8	18%	15		
	Ductal	0	0%	6	13%	6		
	Signal+Ductal	0	0%	31	69%	31		
Total		45	100%	45	100%	90		

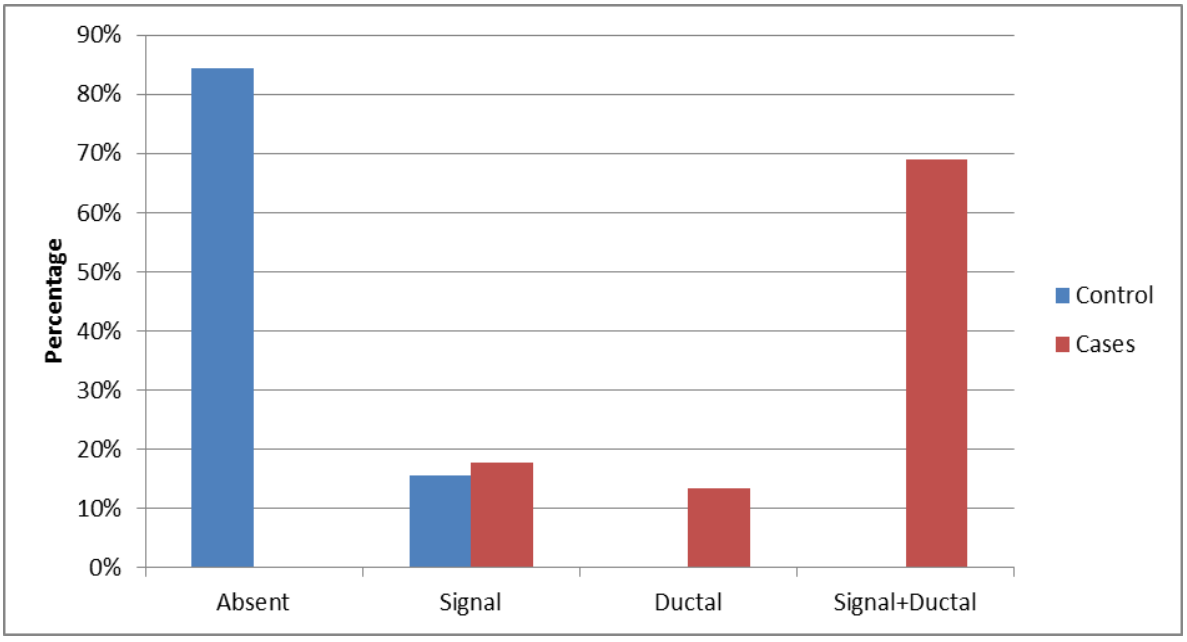


Fig: 22 Both signal as well as ductal changes among cases and controls

Table –18 Percentage of cases and controls showing signal intensity changes

		Group				Total	Chi-square value	p-value
		Control		Cases				
SI	Absent	45	100%	6	13%	51	68.824	0.0001
	Signal	0	0%	39	87%	39		
Total		45	100%	45	100%	90		

Table-19 Sensitivity and specificity of signal intensity changes among cases and controls

Statistic	**Value**	95% CI
Sensitivity	**86.67%**	73.21% to 94.95%
Specificity	**100.00%**	92.13% to 100.00%
Positive Likelihood Ratio		
Negative Likelihood Ratio	**0.13**	0.06 to 0.28
Disease prevalence (*)	**50.00%**	39.27% to 60.73%
Positive Predictive Value (*)	**100.00%**	
Negative Predictive Value (*)	**88.24%**	78.07% to 94.05%
Accuracy (*)	**93.33%**	86.05% to 97.51%

Table-20 Percentage of ductal changes in cases and controls

		Group				Total	Chi-square value	p-value
		Control		Cases				
ductal changes	Absent	45	100%	8	18%	45	62.830	0.0001
	ductal	0	0%	37	82%	45		
Total		45	100%	45	100%	90		

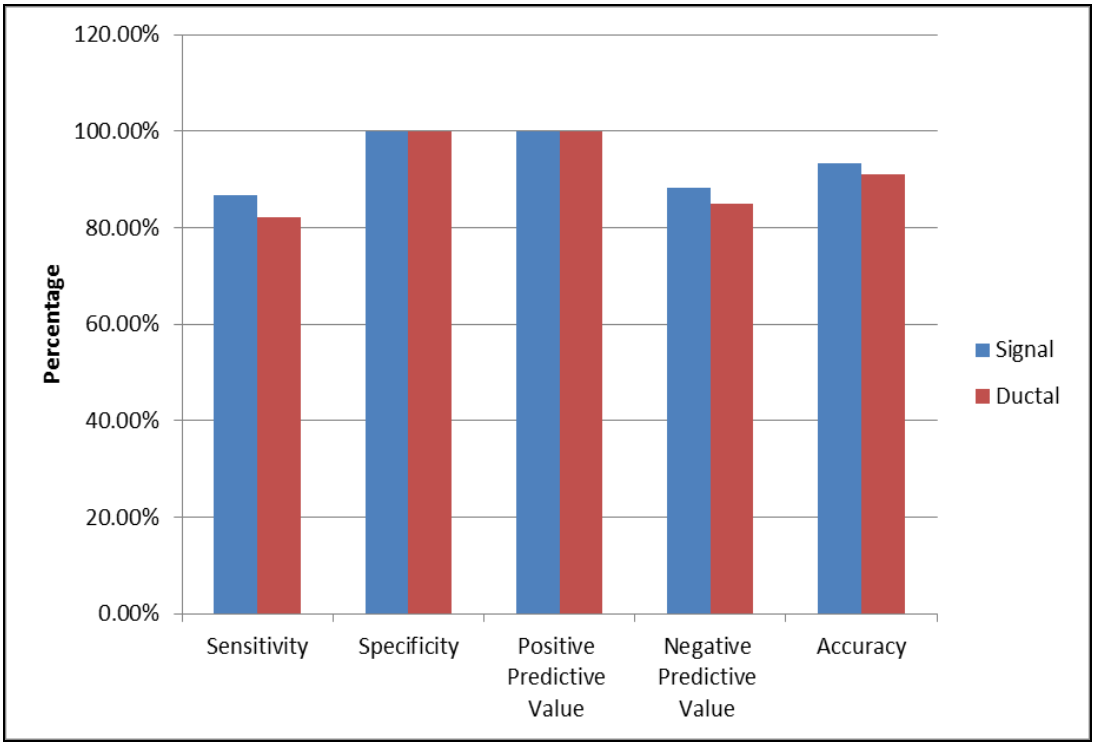


Fig: 23 Sensitivity and specificity of ductal changes in cases and controls

Table –21 Sensitivity and specificity of both signal intensity changes and ductal changes

Statistic	**Value**	95% CI
Sensitivity	**82.22%**	67.95% to 92.00%
Specificity	**100.00%**	92.13% to 100.00%
Positive Likelihood Ratio		
Negative Likelihood Ratio	**0.18**	0.09 to 0.33
Disease prevalence (*)	**50.00%**	39.27% to 60.73%
Positive Predictive Value (*)	**100.00%**	
Negative Predictive Value (*)	**84.91%**	75.00% to 91.34%
Accuracy (*)	**91.11%**	83.23% to 96.08%

Statistic	Signal	Ductal
Sensitivity	**86.67%**	82.2%
Specificity	**100.00%**	100.0%
Positive Predictive Value	**100.00%**	100.0%
Negative Predictive Value	**88.24%**	84.9%
Accuracy	**93.33%**	91.1%

DISCUSSION

Chronic pancreatitis(CP) is characterized by continued inflammation and destruction that leads to irreversible morphological changes in the pancreatic parenchyma & its ductal anatomy.

MRI of the pancreas has undergone a major change due to recent technical improvements, including the capability of providing high-resolution images of the pancreatic duct with the use of half-Fourier T2-weighted pulse sequences. The potential advantage of MRI relative to multidetector CT is its ability to show altered signal intensity in pancreatic disease without the need for contrast material injection. The normal pancreas has been reported to be hyperintense relative to all other solid abdominal tissues on T1-weighted images in multiple studies.[47]

MRI or MR cholangiopancreatography thus provides information on both ductal and parenchymal features of CP, such as T1 signal changes, focal or diffuse gland atrophy, and irregular contour of the pancreatic duct (PD).

Ductal abnormalities are very specific and reliable MRI signs of CP, however, signal intensity changes either by T1-weighted gradient-echo or T1 mapping may precede ductal abnormalities and detect early CP.[66]

It has also been reported that fat saturation or water stimulation accentuates the high SI of the pancreas compared with other abdominal organs by expanding the dynamic range of intraabdominal SI and amplifying the slightly higher SI of the pancreas.[48]

Because of MRI principles, absolute measurements of pancreas SI are not feasible and the use of an internal standard is necessary.

Our study was undertaken to confirm the value of signal intensity evaluation on fat-suppressed T1-weighted sequences and to determine out of liver, spleen and paraspinal muscles which is a reliable standard for detecting a decrease in signal. We also hoped to establish whether accurate results could be achieved by the use of radiologists' interpretations of images and whether calculation from ROI measurement

is necessary to achieve the best results. SIs are cumbersome and are not often used by practising radiologists. The liver, spleen and paraspinal muscles were chosen as internal standards against which to evaluate the pancreas.

Qualitative as well as quantitative analysis of signal intensities was done to evaluate the variation in mean values to derive feasible cut off values that will help in the early diagnosis of chronic pancreatitis. Similarly, the various ductal changes that occur in chronic pancreatitis was assessed to define the role of ductal morphology for better evaluation of chronic pancreatitis and to conclude that out of signal and ductal changes which parameter plays a better role in early diagnosis of chronic pancreatitis.

We did a prospective study on 45 patients diagnosed with chronic pancreatitis on basis of clinical and biochemical parameters. MRCP was performed to assess the signal, morphological and ductal changes.

45 matched controls were also assessed for signal pattern of the pancreas to derive cut off values in patients with chronic pancreatitis.

In our study, there was no significant correlation between the age of the patient, their sex, and the SI of the pancreas. There were 8 women and 37 men with a mean age of 35.53 years $\pm$ 8.56 SD in the normal group. There were 7 women and 38 men with a mean age of 32.33year $\pm$ 12.41 in the pathological group. In our study, the disease was found to be more common among males more so in the younger age group with a mean age of 32.33 years in the pathological group.

Ammann RW et al did a study that the pain in chronic pancreatitis varies among patients. This pattern was illustrated in a prospective cohort of 207 patients with alcoholic CP in which two typical pain patterns were observed. The first was characterized by episodes of pain (usually lasting less than ten days) with pain-free intervals lasting from months to more than a year. The second pattern was characterized by prolonged periods of daily pain or clusters of severe pain exacerbations often requiring repeated hospitalizations.[67]

In another study by Layer P et al, although abdominal pain is the most consistent finding in patients with chronic pancreatitis, it may be absent in some cases. In one series, for example, 20 per cent of patients with chronic pancreatitis presented with evidence of pancreatic exocrine or endocrine dysfunction in the absence of pain.[68]

In our study, out of 45 cases, 38% of patients were presented with pain abdomen predominantly epigastric in location.

Steatorrhea usually occurs before protein deficiencies since lipolytic activity decreases faster than proteolysis.[69] The clinical manifestations of fat malabsorption include loose, greasy, foul-smelling stools that are difficult to flush. Malabsorption of the fat-soluble vitamins (A, D, E, and K) and vitamin B12 may also occur, although clinically symptomatic vitamin deficiency is rare.[70]

Lankisch study included subjects with moderately impaired lipase secretion, and of those patients, two (7.7%) had steatorrhea. Therefore steatorrhea was not limited to those with extreme pancreatic damage.[71]

In our study, out of 45 cases, steatorrhea was present in 22% of patients, 40% of patients were presented with weight loss clinically.

In a previous study, it was mentioned that among pancreatic function tests, faecal elastase measurement is the most sensitive and specific, especially in the early phases of pancreatic insufficiency. According to unpublished data from the manufacturer, values less than 200 mcg/g are suggestive of pancreatic insufficiency (sensitivity and specificity of 93 per cent).[72]

In our study, biochemical parameters such as serum amylase as well as lipase levels were within normal limits. Mean serum amylase and lipase levels were 34.24 ± 6.29 SD and 57.73 ± 12.82 SD in 45 cases respectively. Elastase levels were low (<200) in 39 cases with the mean value of 154.95 ± 20.05 SD. CRP levels were high in both cases as well as controls with the mean value of 9.42 ± 6.30.

In a previous study by Edge MD, ductal dilatation was graded as normal, mild, moderate, or severe. However, like atrophy, grading ducts dilation is also challenging given the lack of widely accepted value for normal and given expected age-related changes. 3 or 3.5 mm has been considered as the threshold by some authors in the past. Irrespective of the diameter (i.e., even if less than 3 mm), an increase in calibre upstream to a stricture or intraductal calculus must be considered dilated.[65]

In another study, the upper range of normal pancreatic duct width was 8.0 mm, 4.0 mm and 2.4 mm in the head, body and tail, respectively. Abnormal ductograms were wider than the normal ranges in the head, body and tail of the pancreas in 14%, 49% and 59% ($p < 0.001$) of the patients, respectively. Establishing normal ranges of the pancreatic duct width may help in the diagnosis of pancreatic disease. Measurements in the body and tail, as compared with the head, are a more sensitive indicator of pancreatic duct dilation.[73]

Axon AT, Classen M et al did a study and concluded that Main duct abnormalities include dilation, narrowing, strictures, filling defects and leaks or cavities. Identification of these abnormalities can be subjective as their definitions are not clear. Average normal main duct diameters were 3.6, 2.7 and 1.6 mm in the head, body and tail respectively, though the upper limited normal may be as high as 6.5, 5 and 3 mm in the head, body and tail, respectively. Main duct dilation is either general (involving more than 2/3 of the main duct) or local (< 2/3 of the ducts). Severe dilation was defined as being > 1 cm.[63]

In our study, Pancreatic duct morphology was maintained in all the controls with 100% sensitivity. However, PD morphology was altered in 45 cases, out of which the mean value of the main pancreatic duct in cases was 5.24 ± 3.51 SD with significant p-value and in controls was 1.64 ± 0.13 SD respectively. Multiple side branches were present in 24% of cases, >3 side branches were present in 42% of cases, <3 side branches were present in 4% cases, no dilated side branches were there

in 29% of cases with a statistically significant p-value with a chi-square value of 49.655.

Soto et al used a respiratory triggered 3D MIP RARE technique to image the pancreatic duct during quiet respiration,21 achieving a sensitivity of 87–100% for duct dilatation, and a sensitivity and specificity of 75% and 69%, respectively, for stricture detection, although the latter was increased to 81% by reviewing the source images themselves.[53]

In our study, Intraductal calcification was documented in 9 out of 45 cases with a sensitivity of 20 % and for ductal dilatation of 82%, Intraductal calcification was absent in 100% of controls.

In our study, Pancreatic duct stricture was present in 56% of cases and was absent in 44% of cases with a statistically significant p-value with a chi-square value of 34.615. Pancreatic duct stricture was absent in 100 % of controls.

So our results were in accordance with the previous study except for intraductal calculus as in our study the sensitivity for detection of intraductal calculus was 20%.

In a previous study, measurement of the pancreatic body was performed at the level of the left margin of the vertebral body with a thickness of 21 mm, 14 mm, and 7 mm serving as the thresholds for varying degrees of atrophy and was graded as </= 7mm : Severe, 7-14 mm: Moderate, 14-21 mm: Mild, 21 mm: Normal.

According to one study by balci et al , 21.8 mm was the mean thickness in patients with a normal pancreatic function test result.[41]

In our study, the mean pancreatic parenchymal thickness was 13.58 mm $\pm$ 5.46 SD in cases and was 22.40 mm $\pm$ 1.21 SD in controls respectively. Our results were in accordance with the previous study

We did qualitative analysis by visually comparing the signal intensities of the pancreas with other organs including the liver, spleen and paraspinal muscles in cases and controls, it was observed that the signal intensity of the pancreas was lowered than liver, spleen in 40 cases which is

considered abnormal and in 5 cases the signal intensity of pancreas was equal to signal intensity of liver which is considered normal. Out of 45 controls, the signal intensity of the pancreas was higher than liver and spleen.

Quantitative analysis was done by comparing signal intensity ratios of the pancreas to other organs in cases and controls.

Benoıt P. Gallix et al did a study to evaluate the accuracy of signal intensity (SI) analysis on unenhanced fat-suppressed T1-weighted MR images in the diagnosis of pancreatic disease and to compare subjective interpretation with different quantitative measurements. They concluded that the pancreas–liver ratio is the best quantitative means of distinguishing normal from the abnormal pancreas. Visual observation by experienced observers (qualitative SI measurement) was just as accurate as a quantitative measurement. Detection of pancreatic pathology can be made with high accuracy by visually comparing the SI of the pancreas with that of the normal liver.[49]

In our study, the mean value of SI of the pancreas in 45 cases was 201.51 ± 93.78 SD with a significant p-value. The mean value of SI of the pancreas in 45 controls was 347.09± 43.71 SD with a significant p-value.

The mean values between cases and controls of pancreas–liver ratios were -0.18 ± 0.19 SD vs. 0.21 ± 0.13 SD, respectively with significant p-value. (SIR P/L-1) at cut off value i.e. 0.00 yielded sensitivity of 86% and specificity of 91% for detection of pancreatic exocrine dysfunction (AUC: 0.87).

The mean values between cases and controls of Pancreas–spleen ratios were -0.05 ± 0.21 SD vs. 0.33 ± 0.11 SD, respectively with significant p-value. (SIR P/S-1) at cut off value i.e. 0.00 yielded sensitivity of 86% and specificity of 56% for detection of pancreatic exocrine dysfunction (AUC: 0.85).

The mean values between cases and controls of Pancreas to paraspinal muscles ratios were 0.02 ± 0.21 SD vs. 0.58 ± 0.22 SD respectively, with

a significant p-value. (SIR P/PSM-1) at cut off value i.e. 0.00 yielded a sensitivity of 88% and specificity of 54% for detection of pancreatic exocrine dysfunction (AUC: 0.86).

So in our study also the qualitative assessment of T1 signal intensity of the pancreas was reduced as compared to other reference organs when observed visually in clinically suspected or biochemically proven cases of chronic pancreatitis. When these findings were correlated quantitatively by comparing the signal intensity ratio of the pancreas to other reference organs (liver, spleen and paraspinal muscles) has comparable performance. The pancreas–liver ratio is the best quantitative means of distinguishing normal from the abnormal pancreas. However, in a few cases the signal intensity of the pancreas was equal to liver qualitatively as well as quantitatively, these were considered as normal. So our results were in accordance with the previous study.

Temel Tirkes, Evan L. Fogel did a study and concluded that there was a significant difference in the signal intensity ratio of the pancreas to the spleen (SIR p/s) between the normal and low bicarbonate groups ($p < 0.0001$). A significant positive correlation was found between pancreatic fluid bicarbonate level and SIR p/s ($p < 0.0001$). SIR p/s of 1.2 yielded sensitivity of 77% and specificity of 83% for detection of pancreatic exocrine dysfunction (AUC: 0.89). T1-weighted MR signal of the pancreas has high sensitivity and specificity for the detection of parenchymal abnormalities related to exocrine dysfunction and can therefore be helpful in the evaluation of suspected early CP.[51]

In our study, there was a significant difference in the signal intensity ratio of the pancreas to the spleen (SIR p/s -1) between cases and controls. The mean values between cases and controls of Pancreas–spleen ratios were -0.05 $\pm$ 0.21 SD vs. 0.33 $\pm$ 0.11 SD, respectively with significant p-value. (SIR P/S-1) at cut off value i.e. 0.00 yielded a sensitivity of 86% and specificity of 56% for detection of pancreatic exocrine dysfunction (AUC: 0.85). But in our study, we found that the pancreas–liver ratio was the best

quantitative means of distinguishing normal from abnormal pancreas with (SIR P/L-1) at cut off value i.e. 0.00 yielded a sensitivity of 86% and specificity of 91% (AUC: 0.87). So our results were not in accordance with the previous study.

Lowered T1 signal intensity (SI) was observed in 87% of patients with Chronic Pancreatitis on qualitative as well as quantitative analysis. Out of 45 cases, 13% showed a similar signal intensity of the pancreas as compared to the signal intensity of the liver. 0ut of 45 controls, 100% showed the higher signal intensity of the pancreas in comparison to other organs. Signal changes (sensitivity 86.67% & specificity 100 %) serve as a more sensitive parameter for chronic pancreatitis than ductal changes(sensitivity 82.2% & specificity 100%)

SUMMARY AND CONCLUSION

1) The present study was conducted at the Department of Radiodiagnosis, Dayanand Medical College and Hospital, Ludhiana.
2) All patients who are diagnosed or suspected to have chronic pancreatitis and are referred for MRI abdomen to the Department of Radiodiagnosis, DMCH Ludhiana for a period of one and half year from 1st Dec 2019 to 31st May 2021.
3) The prospective study was conducted on 45 patients diagnosed with chronic pancreatitis on basis of clinical and biochemical parameters. MRCP was performed to assess the signal, morphological and ductal changes. 45 matched controls were also assessed for signal pattern of the pancreas to derive cut off values in patients with chronic pancreatitis.
4) Mean age was 32.33year $\pm$ 12.417 SD with 38 men and 7 women in the pathological group.
5) The mean values between cases and controls of pancreas–liver ratios were -0.18 + 0.19 SD vs. 0.21 + 0.13 SD, respectively with significant p-value. (SIR P/L-1) at cut off value i.e. 0.00 yielded sensitivity of 86% and specificity of 91% for detection of pancreatic exocrine dysfunction (AUC: 0.87).
6) The mean values between cases and controls of Pancreas–spleen ratios were -0.05 + 0.21 SD vs. 0.33 + 0.11 SD, respectively with significant p-value. (SIR P/S-1) at cut off value i.e. 0.00 yielded sensitivity of 86% and specificity of 56% for detection of pancreatic exocrine dysfunction (AUC: 0.85).
7) The mean values between cases and controls of Pancreas to paraspinal muscles ratios were 0.02 + 0.21 SD vs. 0.58 + 0.22 SD respectively, with a significant p-value. (SIR P/PSM-1) at cut off

value i.e. 0.00 yielded a sensitivity of 88% and specificity of 54% for detection of pancreatic exocrine dysfunction (AUC: 0.86).

8) PD morphology was altered in 45 cases, out of which the mean value of the main pancreatic duct in cases was 5.24 + 3.51 SD with significant p-value and in controls was 1.64 + 0.13 SD respectively.
9) Multiple side branches were present in 24% of cases, >3 side branches were present in 42% of cases, <3 side branches were present in 4% cases, no dilated side branches were there in 29% of cases with a statistically significant p-value with a chi-square value of 49.655.
10) Intraductal calcification was documented in 9 out of 45 cases with a sensitivity of 20 % and for ductal dilatation of 82%, Intraductal calcification was absent in 100% of controls.
11) Pancreatic duct stricture was present in 56% of cases and was absent in 44% of cases with a statistically significant p-value with a chi-square value of 34.615. Pancreatic duct stricture was absent in 100 % of controls.
12) The mean pancreatic parenchymal thickness was 13.58 mm $\pm$ 5.46 SD in cases and was 22.40 mm $\pm$ 1.21 SD in controls respectively
13) Out of 45 controls, 100% showed the higher signal intensity of the pancreas in comparison to other organs. Signal changes (sensitivity 86.67% & specificity 100 %) serve as a more sensitive parameter for chronic pancreatitis than ductal changes(sensitivity 82.2% & specificity 100%)
14) The pancreas–liver ratio is the best quantitative means of distinguishing normal from the abnormal pancreas. Signal changes (sensitivity 86.67% & specificity 100 %) serve as a more sensitive parameter for chronic pancreatitis than ductal changes(sensitivity 82.2% & specificity 100%) for early diagnosis of chronic pancreatitis.

CONCLUSION

Visual analysis (Qualitative) of the pancreatic signal pattern is a dependable parameter for distinguishing normal from the abnormal pancreas. Quantitative analysis using signal intensity patterns has comparable performance. The pancreas–liver ratio is the best quantitative means of distinguishing normal from the abnormal pancreas. Signal changes (sensitivity 86.67% & specificity 100%) serve as a more sensitive parameter for chronic pancreatitis than ductal changes(sensitivity 82.2% & specificity 100%) for early diagnosis of chronic pancreatitis.

BIBLIOGRAPHY

1. Quencer K, Kambadakone A, Sahani D, Guimaraes AS. Imaging of the pancreas: part 1. Appl Radiol. 2013;42:14-20.
2. Leyendecker JR, Oliphant M. Pancreas. In: Dalrymple NC, Leyendecker JR, Oliphant M, eds. Problem solving in abdominal imaging. Philadelphia, PA: Mosby Elsevier; 2009
3. Semelka RC, Ascher SM. MR imaging of the pancreas. Radiology 1993; 188:593–602.
4. Heyn C, Sue-Chue-Lam D, Jhaveri K, Haider MA. MRI of the pancreas: problem solving tool. Journal of Magnetic Resonance Imaging. 2012;36:1037-51.
5. Vitellas KM, Keogan MT, Spritzer CE, Nelson RC. MR cholangiopancreatography of bile and pancreatic duct abnormalities with emphasis on the single-shot fast spin-echo technique. Radiographics. 2000;20:939-57.
6. Mortelé KJ, Rocha TC, Streeter JL, Taylor AJ. Multimodality imaging of pancreatic and biliary congenital anomalies. *Radiographics.* 2006;26:715-31.
7. Matsumoto Y, Fujii H, Itakura J. Pancreaticobiliary maljunction: pathophysiological and clinical aspects and the impact on biliary carcinogenesis. Langenbecks Arch Surg. 2003;388(2):122-131.
8. Kozu T, Suda K, Toki F. Pancreatic development and anatomical variation. Gastrointest Endosc Clin N Am. 1995;5:1-30.
9. Wang DB, Yu J, Fulcher AS, Turner MA. Pancreatitis in patients with pancreas divisum: imaging features at MRI and MRCP. World Journal of Gastroenterology: WJG. 2013;19:4907.
10. Gerke H, Byrne MF, Stiffler HL, Obando JV, Mitchell RM, Jowell PS, et al. Outcome of endoscopic minor papillotomy in patients with symptomatic pancreas divisum. JOP. 2004;5:122-31.

11. Kamat R, Gupta P, Rana S. Imaging in chronic pancreatitis: state of the art review. The Indian journal of radiology & imaging. 2019;29:201.
12. Perez-Johnston R, Sainani NI, Sahani DV. Imaging of chronic pancreatitis (including groove and autoimmune pancreatitis). Radiologic Clinics. 2012;50:447-66.
13. Sharma R, Kandasamy D. Imaging in chronic pancreatitis. Pancreapedia: The Exocrine Pancreas Knowledge Base. 2015.
14. Siddiqi AJ, Miller F. Chronic pancreatitis: Ultrasound, computed tomography, and magnetic resonance imaging features. Semin Ultrasound CT MRI. 2007;28:384-94.
15. Issa Y, Kempeneers MA, van Santvoort HC, Bollen TL, Bipat S, et al. Diagnostic performance of imaging modalities in chronic pancreatitis: A systematic review and meta-analysis. Eur Radiol. 2017;27:3820-44.
16. Choueiri NE, Balci NC, Alkaade S, Burton FR. Advanced imaging of chronic pancreatitis. Curr Gastroenterol Rep. 2010;12:114-20.
17. Graziani R, Tapparelli M, Malagò R, Girardi V, Frulloni L, Cavallini G, et al. The various imaging aspects of chronic pancreatitis. JOP. 2005;6:73-88.
18. Sharma R, Kandasamy D. Imaging in chronic pancreatitis. Pancreapedia: The Exocrine Pancreas Knowledge Base. 2015
19. Laterza L, Scaldaferri F, Bruno G, Agnes A, Boškoski I, Ianiro G, et al. Pancreatic function assessment. Eur Rev Med Pharmacol Sci. 2013;17:65-71.
20. Siddiqi AJ, Miller F. Chronic pancreatitis: Ultrasound, computed tomography, and magnetic resonance imaging features. Semin Ultrasound CT MRI. 2007;28:384-94.
21. Dimcevski G, Erchinger FG, Havre R, Gilja OH. Ultrasonography in diagnosing chronic pancreatitis: New aspects. *World J Gastroenterol.* 2013;19:7247–57
22. Elmas N. The role of diagnostic radiology in pancreatitis. *Eur J Radiol.* 2001;38:120–32

23. Laterza L, Scaldaferri F, Bruno G, Agnes A, Boškoski I, Ianiro G, et al. Pancreatic function assessment. *Eur Rev Med Pharmacol Sci.* 2013;17:65-71.
24. Alpern MB, Sandler MA, Kellman GM, Madrazo BL. Chronic pancreatitis: Ultrasonic features. *Radiology.* 1985;155:215-9.
25. Ammann RW, Muench R, Otto R, Buehler H, Freiburghaus AU, Siegenthaler W. Evolution and regression of pancreatic calcification in chronic pancreatitis: A prospective long-term study of 107 patients. *Gastroenterology.* 1988;95:1018–28.
26. Siddiqi AJ, Miller F. Chronic pancreatitis: Ultrasound, computed tomography, and magnetic resonance imaging features. *Semin Ultrasound CT MRI.* 2007;28:384-94.
27. Raman SP, Fishman EK, Lennon AM. Endoscopic ultrasound and pancreatic applications: What the radiologist needs to know. *Abdom Imaging.* 2013;38:1360-72.
28. Guo J, Sun S. Endoscopic ultrasound for the diagnosis of chronic pancreatitis. *Pancreapedia Exocrine Pancreas Knowl Base.* 2015.
29. Iglesias-Garcia J, Lindkvist B, Lariño-Noia J, Domínguez-Muñoz JE. Endoscopic ultrasound elastography. *Endosc Ultrasound.* 2012;1:8-16.
30. Pozzi R, Parzanese I, Baccarin A, Giunta M, Conti CB, Cantù P, et al. Point shear-wave elastography in chronic pancreatitis: A promising tool for staging disease severity. *Pancreatology.* 2017;17:905-10.
31. Mateen MA, Muheet KA, Mohan RJ, Rao PN, Majaz HM, Rao GV, et al. Evaluation of ultrasound based acoustic radiation force impulse (ARFI) and eSie touch sonoelastography for diagnosis of inflammatory pancreatic diseases. *JOP.* 2012;13:36-44.
32. Domínguez-Muñoz JE, Alvarez-Castro A, Lariño-Noia J, Nieto L, Iglesias-García J. Endoscopic ultrasonography of the pancreas as an indirect method to predict pancreatic exocrine insufficiency in patients with chronic pancreatitis. *Pancreas.* 2012;41:724-8.

33. Soto JA, Barish MA, Yucel EK, Clarke P, Siegenberg D, Chuttani R, et al. Pancreatic duct: MR cholangiopancreatography with a three-dimensional fast spin-echo technique. *Radiology.* 1995;196:459–64
34. Choueiri NE, Balci NC, Alkaade S, Burton FR. Advanced imaging of chronic pancreatitis. *Curr Gastroenterol Rep.* 2010;12:114–20
35. Frulloni L, Castellani C, Bovo P, Vaona B, Calore B, Liani C, et al. Natural history of pancreatitis associated with cystic fibrosis gene mutations. *Dig Liver Dis.* 2003;35:179–85
36. Sinha A, Singh VK, Cruise M, Afghani E, Matsukuma K, Ali S, et al. Abdominal CT predictors of fibrosis in patients with chronic pancreatitis undergoing surgery. *Eur Radiol.* 2015;25:1339–46
37. Kim DH, Pickhardt PJ. Radiologic assessment of acute and chronic pancreatitis. *Surg Clin North Am.* 2007;87:1341–58
38. Tirkes T, Fogel EL, Sherman S, et al. Detection of exocrine dysfunction by MRI in patients with early chronic pancreatitis. Abdom Radiol (NY) 2017;42:544–551.
39. Balci NC, Smith A, Momtahen AJ. MRI and S-MRCP findings in patients with suspected chronic pancreatitis: correlation with endoscopic pancreatic function testing (ePFT). J Magn Reson Imaging 2010;31:601–606
40. Tirkes T, Lin C, Cui E. Quantitative MR evaluation of chronic pancreatitis: extracellular volume fraction and MR relaxometry. AJR Am J Roentgenol 2018;210:533–542
41. Balci NC, Smith A, Momtahen AJ. MRI and S-MRCP findings in patients with suspected chronic pancreatitis: correlation with endoscopic pancreatic function testing (ePFT). J Magn Reson Imaging 2010;31:601–606.
42. Carbognin G, Pinali L, Girardi V, Casarin A, Mansueto G, Mucelli RP. Collateral branches IPMTs: secretin-enhanced MRCP. Abdom Imaging 2007;32(3):374-80.

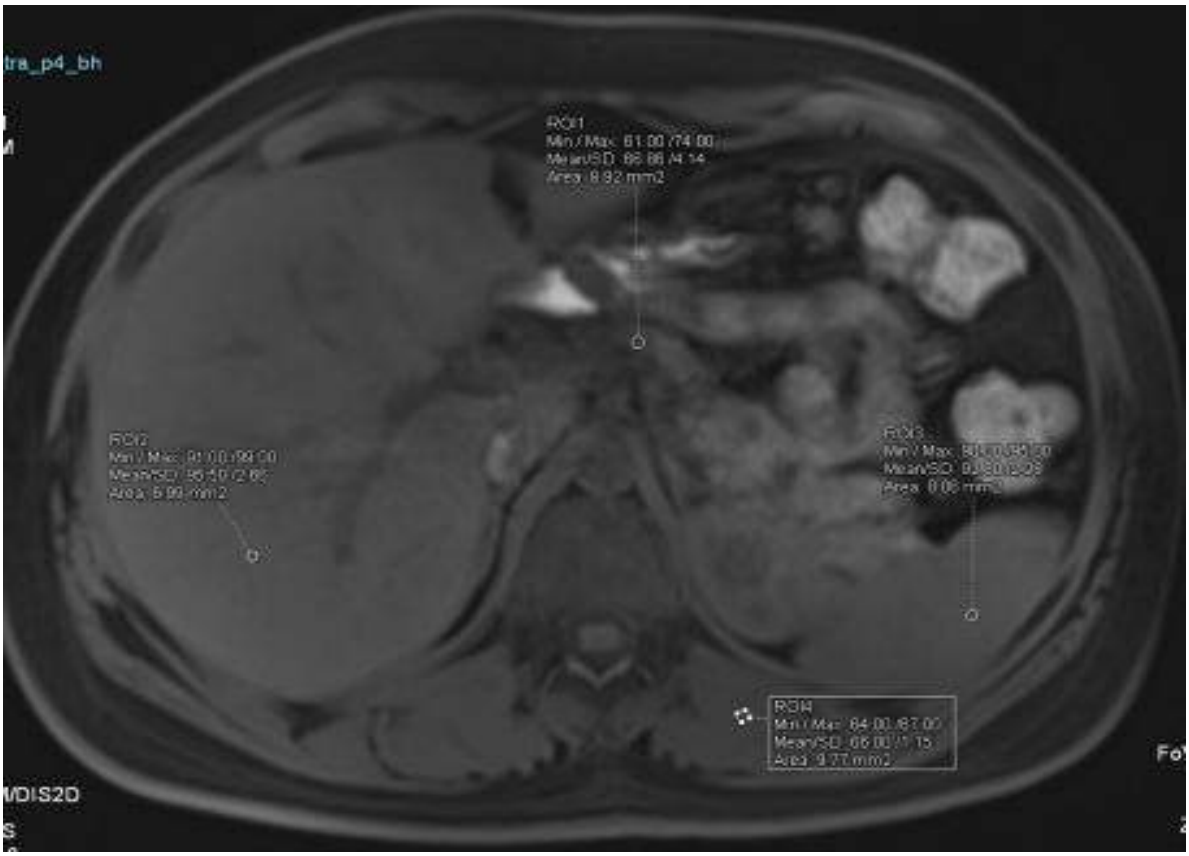
43. Takehara Y, Ichijo K, Tooyama N. *Breath-hold MR cholangiopancreatography with a long-echo-train fast spin-echo sequence and a surface coil in chronic pancreatitis.* Radiology. 1994;192:73-8.
44. Masui T, Takehara Y, Ichijo K. *Evaluation of pancreatic diseases: comparison of single thick slice projection MRCP and multiple thin slice MPR MRCP (abstr).* Radiology 1997;205:504.
45. Fulcher AS, Turner MA, Capps GW. *Half-Fourier RARE MR cholangiopancreatography in 300 subjects.* Radiology. 1998;207:21-32.
46. Bosmans H, Van Hoe L, Gryspeerdt S. *Single-shot T2 weighted MR imaging of the upper abdomen: preliminary experience with the double echo HASTE technique.* Am J Roentgenol 1997;169:1291-3.
47. Mitchell DG, Winston CB, Outwater EK, Ehrlich SM. *Delineation of pancreas with MR imaging: multiobserver comparison of five pulse sequences.* JMRI 1995;5:193-9.
48. Winston CB, Mitchell DG, Outwater EK, Ehrlich SM. Pancreatic signal intensity on T1-weighted fat saturation MR images: clinical correlation. J Magn Reson Imaging. 1995;5:267-71.
49. Gallix BP, Bret PM, Atri M, Lecesne R, Reinhold C. Comparison of Qualitative and Quantitative Measurements on Unenhanced T1-Weighted Fat Saturation MR Images in Predicting Pancreatic. JMagn Reson Imaging. 2005;21:583-9.
50. Dasyam AK, Shah ZK, Tirkes T, Dasyam N, Borhani AA. Cross-sectional imaging-based severity scoring of chronic pancreatitis: why it is necessary and how it can be done. Abdominal Radiology. 2020;45:1447-57.
51. Tirkes T, Fogel EL, Sherman S, Lin C, Swensson J, Akisik F, Sandrasegaran K. Detection of exocrine dysfunction by MRI in patients with early chronic pancreatitis. Abdominal Radiology. 2017;42:544-51.

52. Hekimoglu K, Ustundag Y, Dusak A, Erdem Z, Karademir B, Aydemir S, et al. MRCP vs. ERCP in the evaluation of biliary pathologies: review of current literature. J Dig Dis. 2008;9:162-9.
53. Soto JA, Barish MA, Yucel EK, Clarke P, Siegenberg D, Chuttani R, et al. Pancreatic duct: MR cholangiopancreatography with a three-dimensional fast spin-echo technique. Radiology. 1995;196:459-64.
54. Calvo MM, Calderón A, Heras I, Durán M, Orive V, Cabriada J, et al. Magnetic resonance study of the pancreatic duct. Rev Esp Enferm Dig. 1999;91:287-96.
55. Lomanto D, Pavone P, Laghi A, Panebianco V, Mazzocchi P,Fiocca F, et al. Magnetic resonance-cholangiopancreatography in the diagnosis of biliopancreatic diseases. Am J Surg. 1997;174:33-8.
56. Tirkes T, Lin C, Fogel EL, Sherman SS, Wang Q, Sandrasegaran K. T1 mapping for diagnosis of mild chronic pancreatitis. Journal of Magnetic Resonance Imaging. 2017;45:1171-6.
57. Srinivasan S, Chatterjee A. Recent Advances in Pancreatic MR Imaging: A Guide on How, When, and Why to Perform. Journal of Gastrointestinal and Abdominal Radiology. 2020;3:002-13.
58. Manikkavasakar S, AlObaidy M, Busireddy KK, Ramalho M, Nilmini V, Alagiyawanna M, et al. Magnetic resonance imaging of pancreatitis: An update. *World J Gastroenterol*. 2014;20:14760-77.
59. Tirkes T. Chronic pancreatitis: what the clinician wants to know from MR imaging. Magnetic Resonance Imaging Clinics. 2018;26:451-61.
60. Alkaade S, Balci NC, Momtahen AJ, Burton F. Normal pancreatic exocrine function does not exclude MRI/MRCP chronic pancreatitis findings. Journal of clinical gastroenterology. 2008;42:950-5.
61. Tirkes T, Lin C, Cui E, Deng Y, Territo PR, Sandrasegaran K, et al. Quantitative MR evaluation of chronic pancreatitis: extracellular volume fraction and MR relaxometry. AJR. American journal of roentgenology. 2018;210:533.

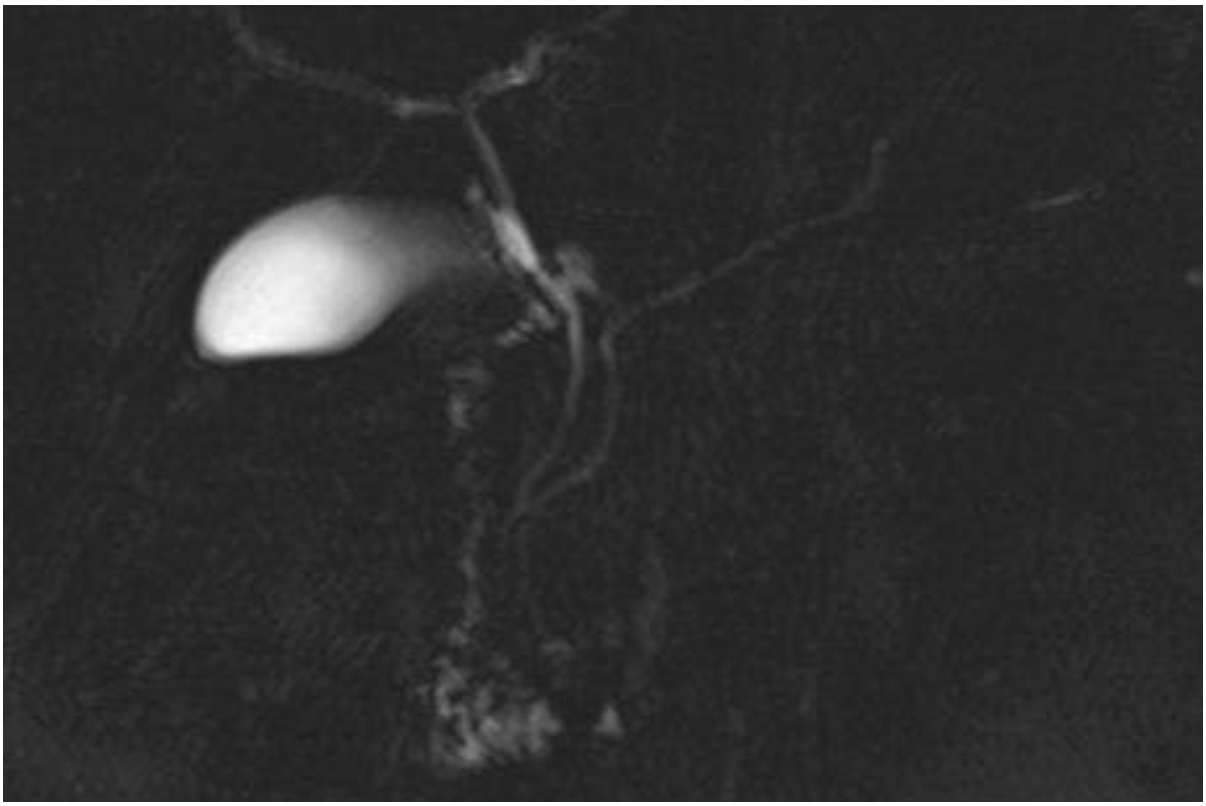
62. Kamat R, Gupta P, Rana S. Imaging in chronic pancreatitis: state of the art review. The Indian journal of radiology & imaging. 2019;29:201.
63. Axon AT, Classen M, Cotton PB. Pancreatography in chronic pancreatitis: international definitions. *Gut.* 1984;25:1107–12
64. Cheng M, Gromski MA, Fogel EL, DeWitt JM, Patel AA, Tirkes T. T1 mapping for the diagnosis of early chronic pancreatitis: correlation with Cambridge classification system. The British Journal of Radiology. 2021;94:20200685.
65. Edge MD, Hoteit M, Patel AP, Wang X, Baumgarten DA, Cai Q. Clinical significance of main pancreatic duct dilation on computed tomography: single and double duct dilation. World Journal of Gastroenterology: WJG. 2007;13:1701.
66. Tirkes T, Lin C, Fogel EL, Sherman SS, Wang Q, Sandrasegaran K. T1 mapping for diagnosis of mild chronic pancreatitis. *J Magn Reson Imaging*. 2017;45:1171-6.
67. Ammann RW, Muellhaupt B. The natural history of pain in alcoholic chronic pancreatitis. *Gastroenterology*. 1999;116:1132-40.
68. Layer P, Yamamoto H, Kalthoff L, Clain JE, Bakken LJ, DiMagno EP. The different courses of early- and late-onset idiopathic and alcoholic chronic pancreatitis. *Gastroenterology* . 1994;107:1481.
69. Mergener K, Baillie J. Chronic pancreatitis. Lancet 1997;350:1379-85.
70. Toskes PP, Hansell J, Cerda J, Deren JJ. Vitamin B 12 malabsorption in chronic pancreatic insufficiency. N Engl J Med. 1971;284:627-32.
71. Lankisch PG, Lembcke B, Wemken G, Creutzfeldt W. Functional reserve capacity of the exocrine pancreas. Digestion. 1986;35:175-81.
72. Keim V, Teich N, Moessner J. Clinical value of a new fecal elastase test for detection of chronic pancreatitis. Clin Lab. 2003;49:209-15.
73. Ladas SD, Tassios PS, Giorgiotis K, Rokkas T, Theodosiou P, Raptis SA. Pancreatic duct width: its significance as a diagnostic criterion for pancreatic disease. Hepato-gastroenterology. 1993;40:52-5.

Picture 1) A patient (No; 11 as in the masterchart) presented with epigastric pain and was underwent MRCP. This is T1 Weighted fat sat image which showed Mean Signal intensity of pancreas by placing ROI i.e. (48.00 ±1.83 SD), Liver (93.50± 0.84 SD), Spleen (70.00 ± 0.82 SD) and paraspinal muscles (64.50 ±1.29)respectively)

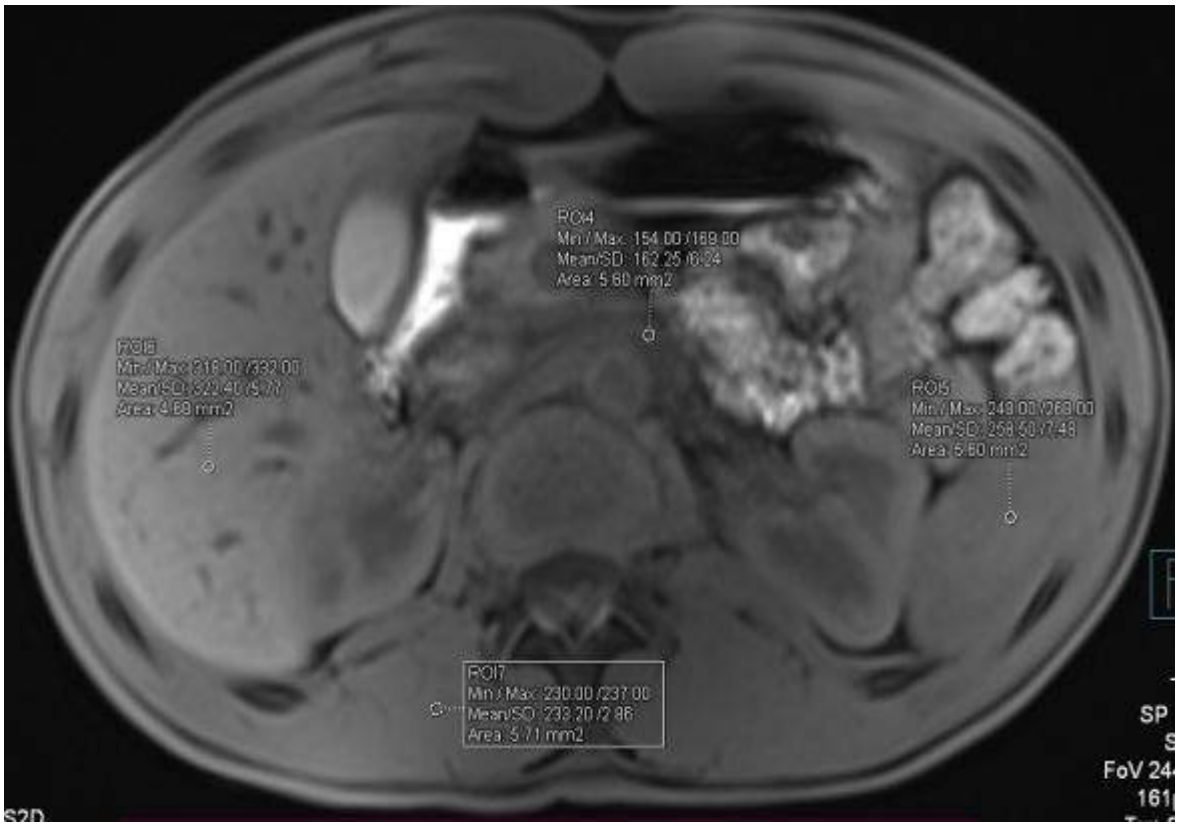
Picture 2) MIP image (No; 11 as in the masterchart) showed the normal ductal morphology in the same patient .On the basis of diffuse pancreatic parenchymal signal changes only, the patient was labeled as chronic pancreatitis and was further put on medical management and on follow up improved clinically.



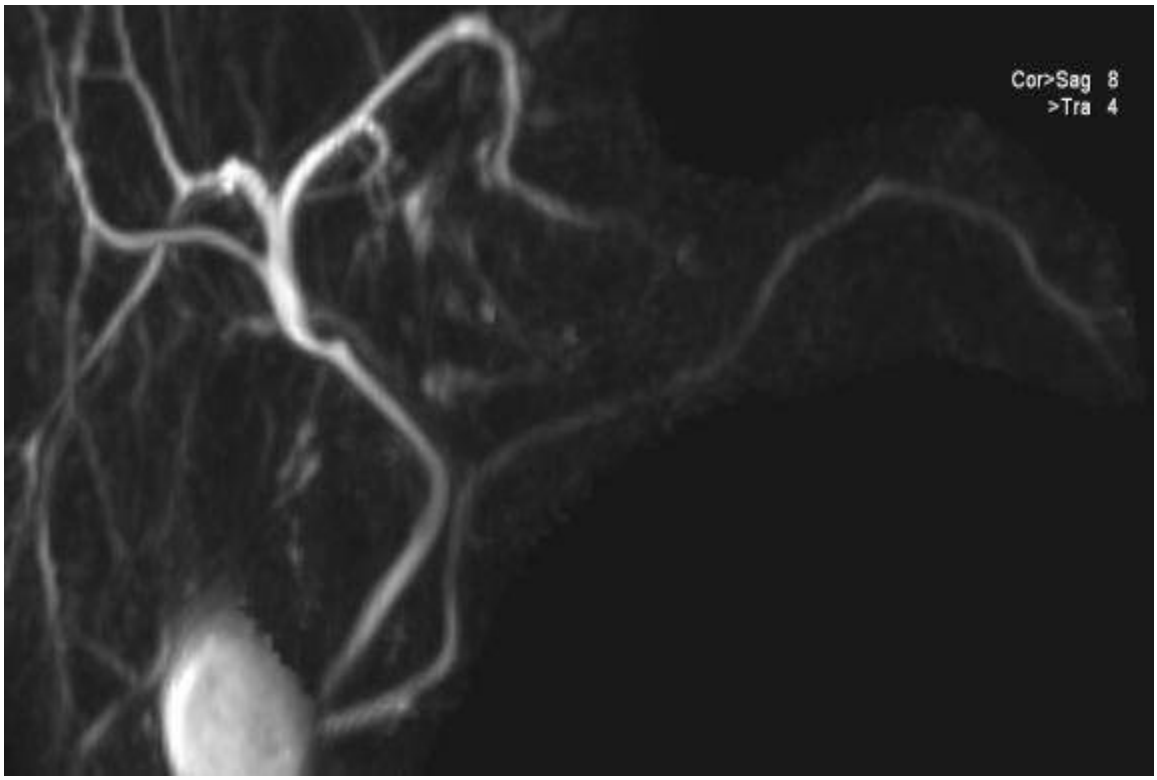
Picture 3) (No; 23 in masterchart) A patient presented with epigastric pain and was underwent MRCP. This is T1 weighted fat sat image which showed the mean signal intensity of pancreas by placing ROI i.e. (66.86 $\pm$ 4.14 SD), Liver (95.50 $\pm$ 2.66 SD), Spleen (92. 80 $\pm$ 2.28 SD), Paraspinal muscles (66.00 $\pm$ 1.15 SD).



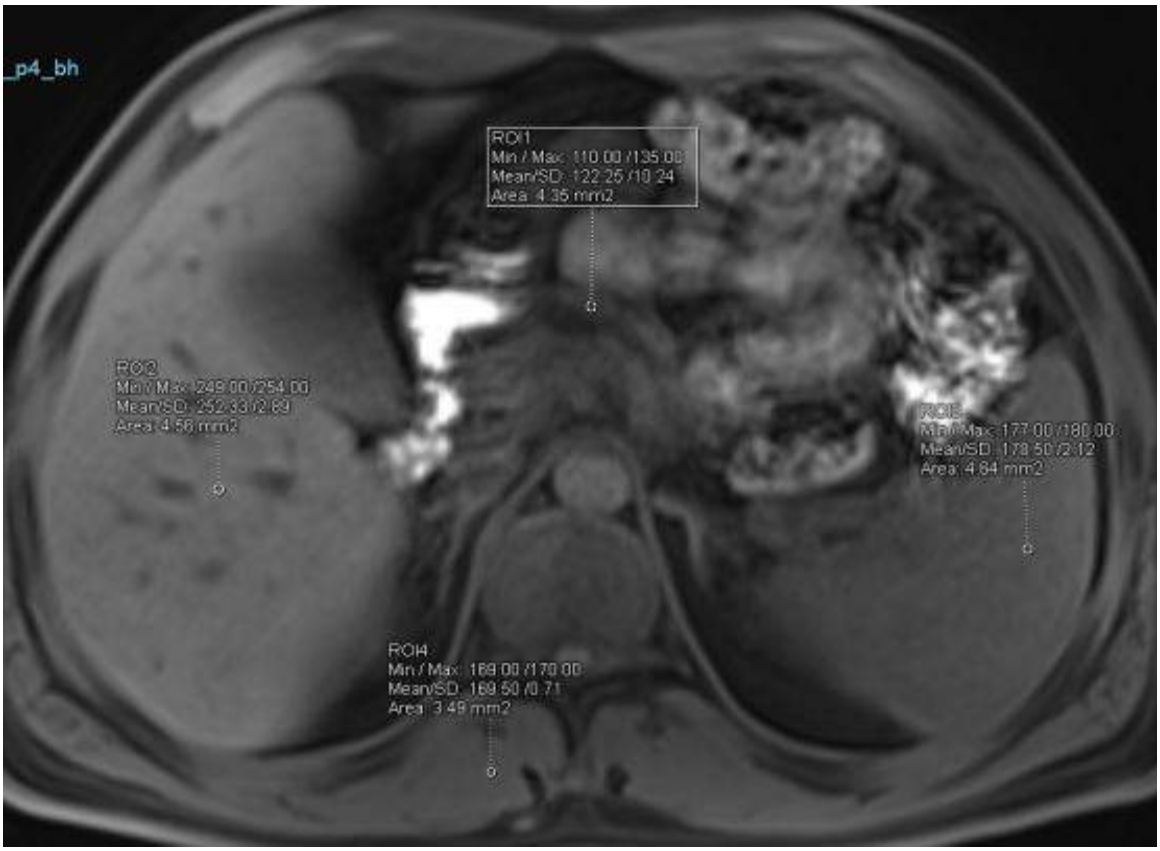
Picture 4)(No; 23 in masterchart) MIP image in the same patient revealed the normal ductal morphology. The patient was labeled as case of chronic pancreatitis on the basis of reduced pancreatic parenchymal signal and was put on medical treatment and further improved clinically.



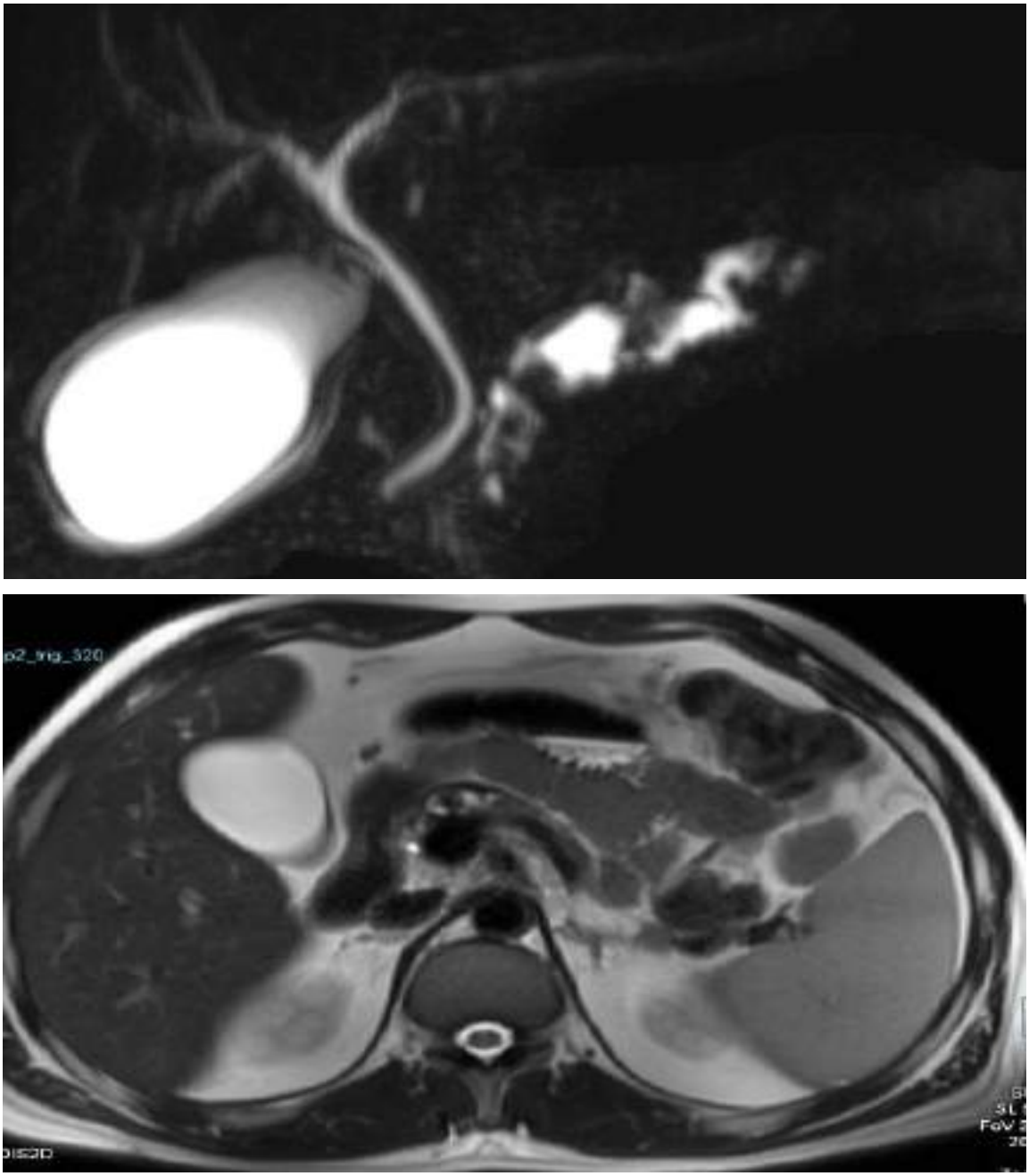
Picture 5) (No; 35 in masterchart) A patient presented with epigastric pain and was underwent MRCP. This is T1 weighted fat sat image which showed the mean signal intensity of pancreas by placing ROI i.e. (162.25.86 $\pm$ 6.24 SD), Liver (322.40 $\pm$ 5.77 SD), Spleen (259.50 $\pm$ 7.48 SD), Paraspinal muscles (233.20 $\pm$ 2.86 SD).



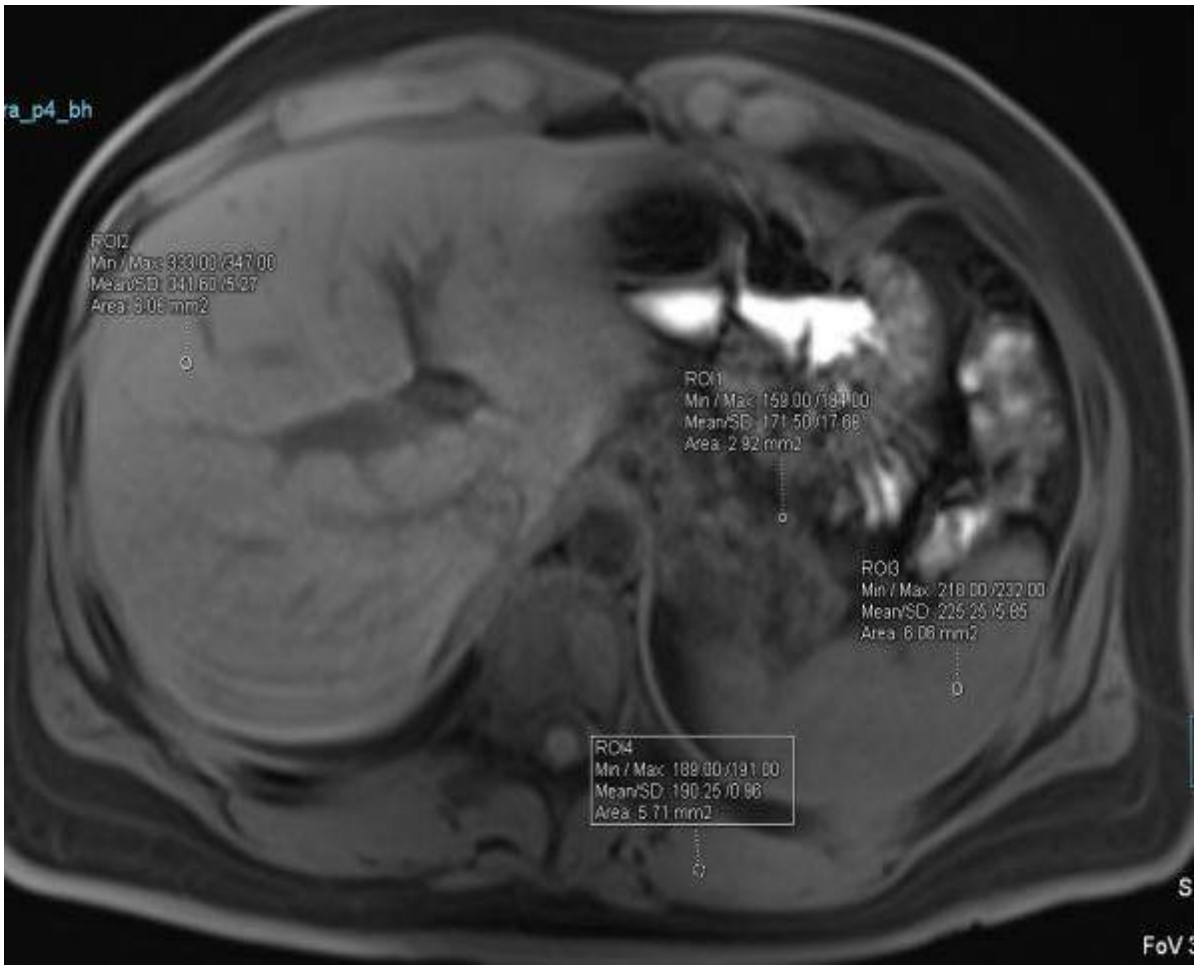
Picture: 6) (No; 35 in masterchart) MIP image in the same patient revealed the normal ductal morphology. The patient was labeled as case of chronic pancreatitis on the basis of reduced pancreatic parenchymal signal and was put on medical treatment and further improved clinically.



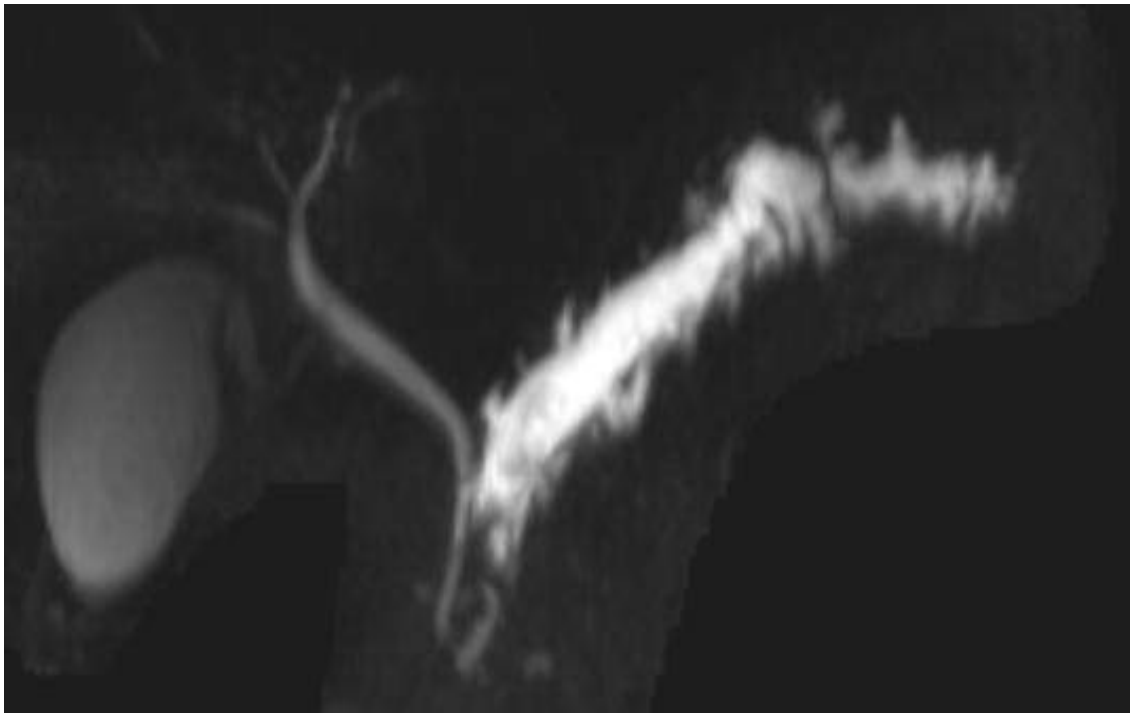
Picture 7) A (No; 24 as in the masterchart) patient presented with steatorrhea & weight loss and was underwent MRCP. This is T1 weighted fat sat image which showed the mean signal intensity of pancreas by placing ROI i.e. (122.25 $\pm$ 10.24 SD), Liver (252.33 $\pm$ 2.89 SD), Spleen (178.50 $\pm$ 2.12 SD), Paraspinal muscles (169.50 $\pm$ 0.71 SD).



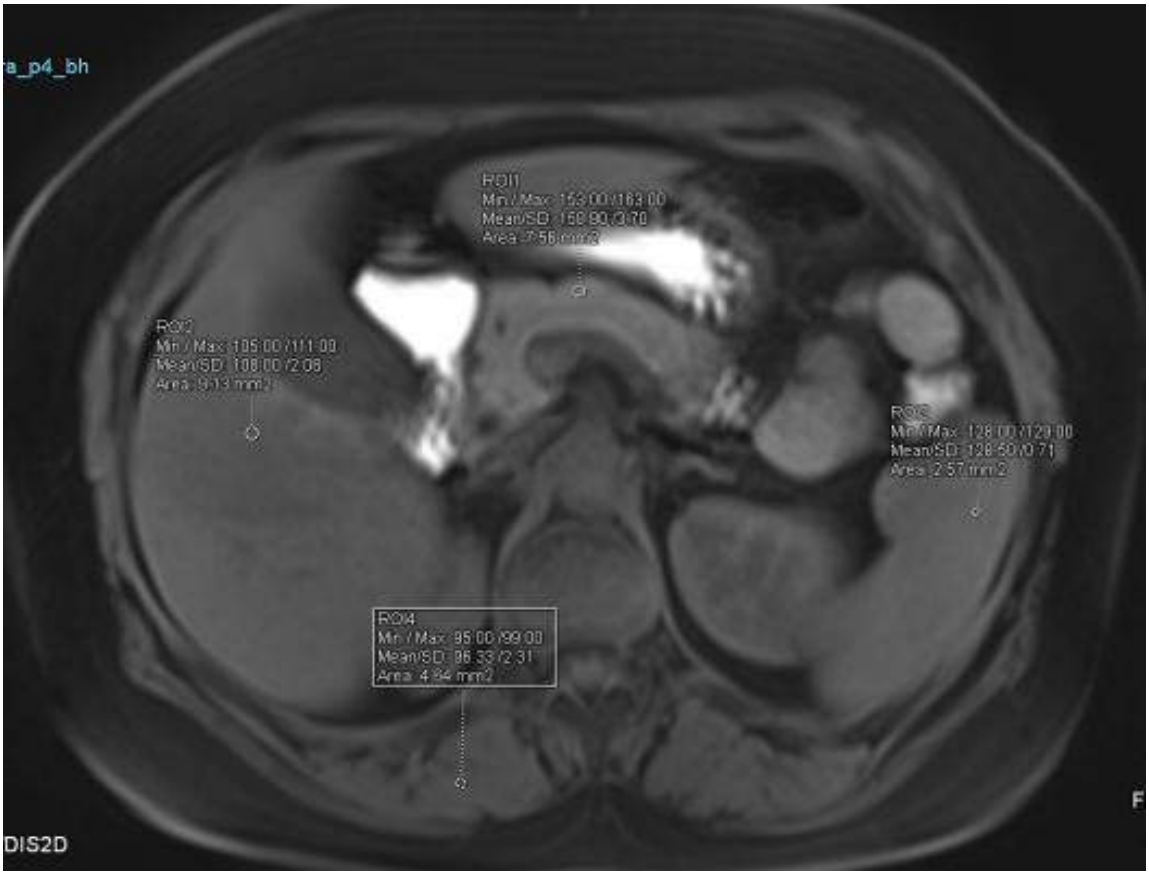
Picture 8, 9) (No; 24 as in the masterchart) MIP & Axial T2 haste image in the same patient revealed MPD which appeared markedly dilated and irregular in calibre. There was sudden change in caliber noted in the region of neck s/o dominant stricture. Multiple signal voids were present within its lumen in keeping with calculi. The patient was labeled as case of severe chronic calcific pancreatitis with dominant stricture in neck with multiple intraductal calculi and was underwent PD stenting and further improved clinically.



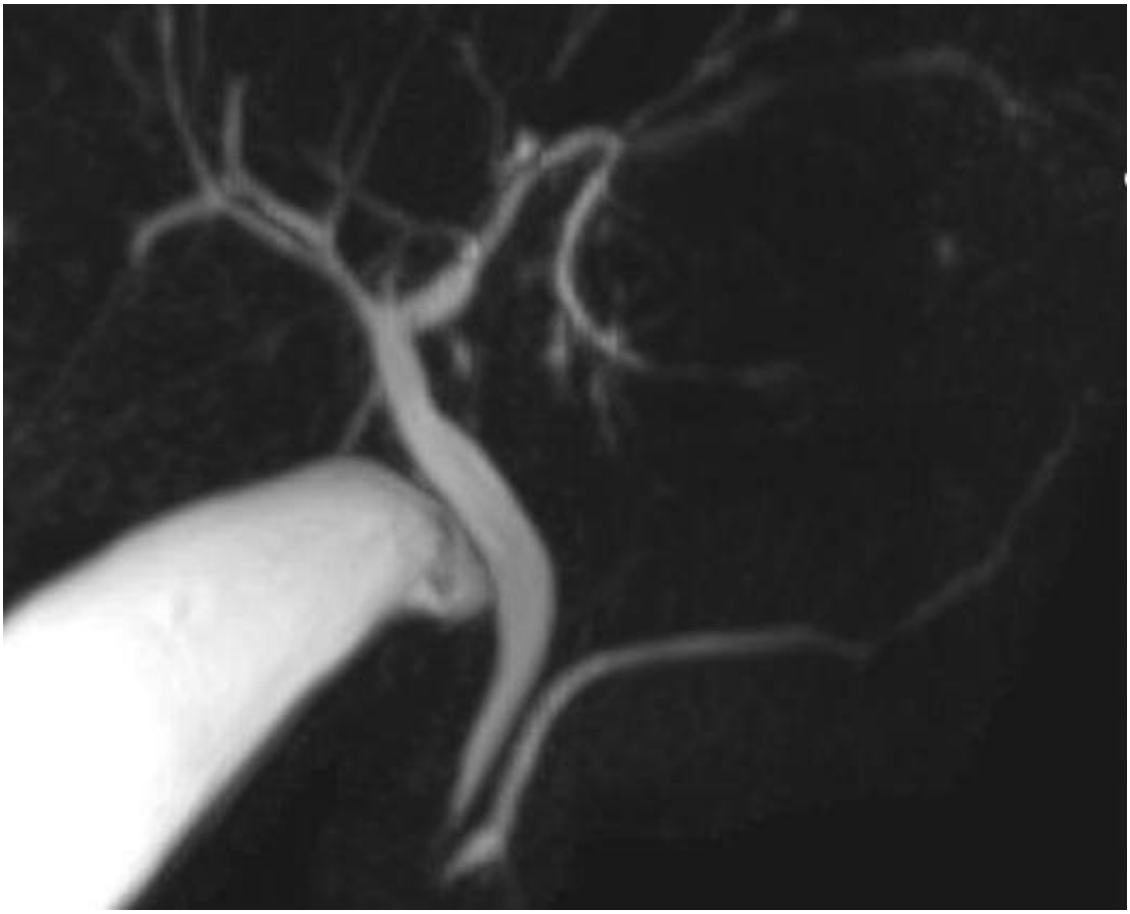
Picture 10) (No; 14 as in the masterchart) A patient presented with steatorrhea & weight loss and was underwent MRCP. This is T1 weighted fat sat image which showed the mean signal intensity of pancreas by placing ROI i.e. (171.50 $\pm$ 17.68 SD), Liver (341.60 $\pm$ 5.27 SD), Spleen (225.25 $\pm$ 5.85 SD), Paraspinal muscles (190.25 $\pm$ 0.96 SD).



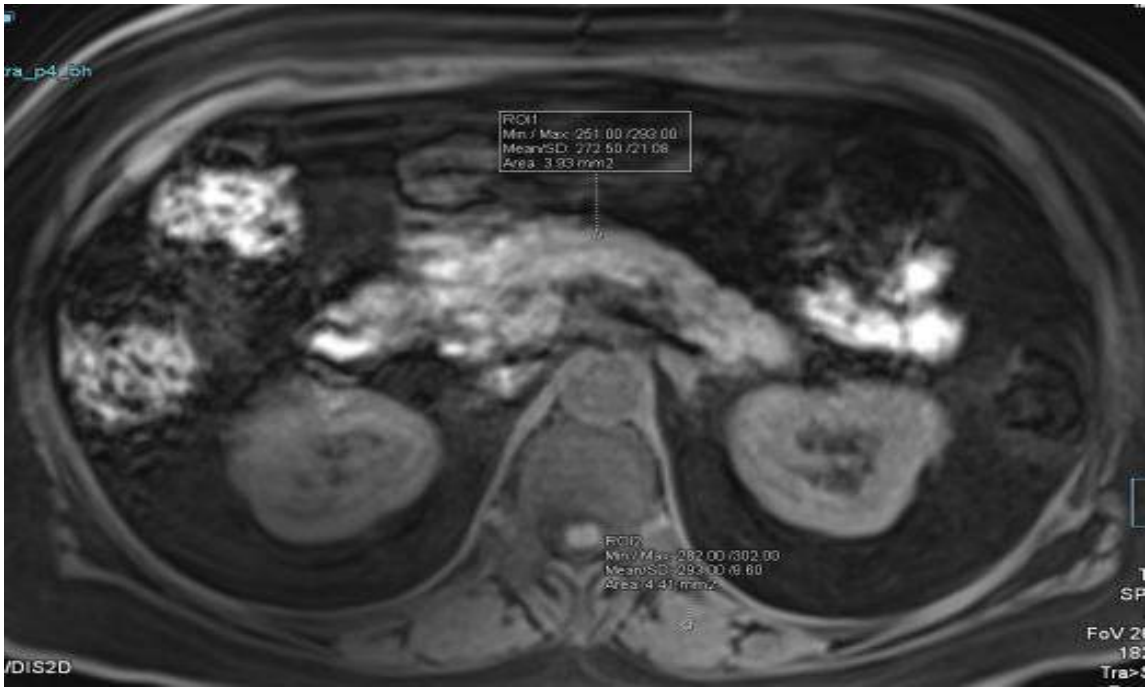
Picture 11) MIP (No; 14 as in the masterchart) image of above mentioned patient revealed markedly dilated MPD with contour irregularity and multiple side duct branches. Two signal voids were noted in MPD s/o calculi beyond which duct was normal in calibre in keeping with stricture. Patient was labeled as case of chronic calcific pancreatitis with dominant stricture and calculi in head and was underwent PD stenting and improved clinically

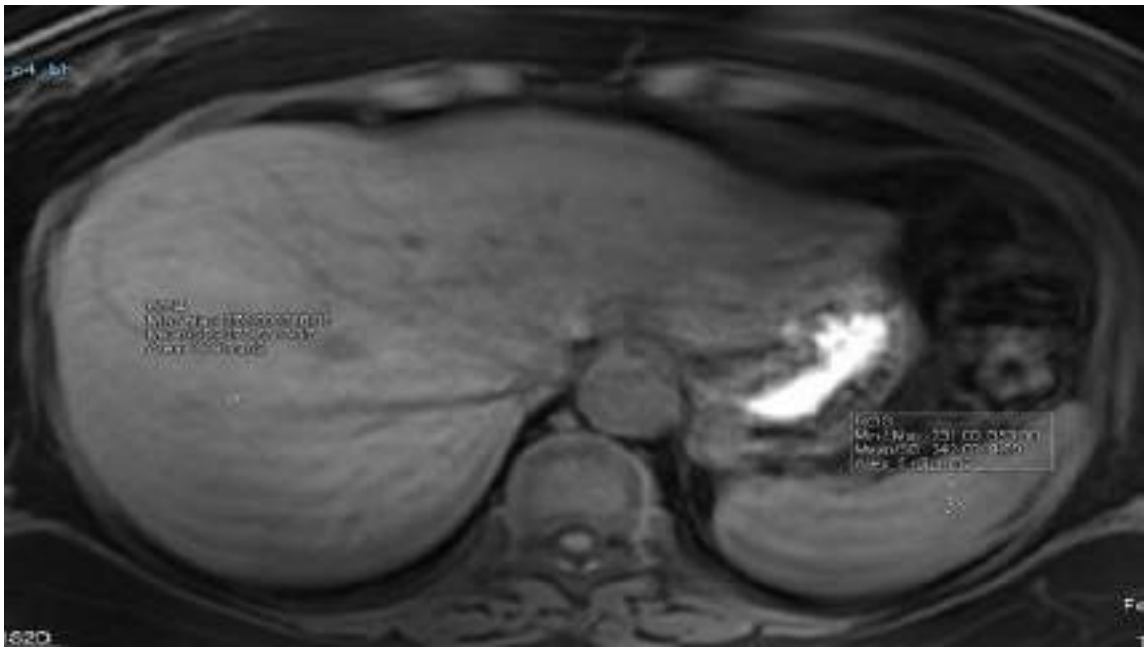


Picture 12) (No; 75 as in the masterchart) A patient presented with fever & vomiting and was underwent MRCP to rule out choledocolithiasis. This is T1 weighted fat sat image which showed the mean signal intensity of pancreas by placing ROI i.e. (158.80 $\pm$ 3.70 SD), Liver (108.00 $\pm$ 2.08 SD), Spleen (128.50 $\pm$ 0.71 SD), Paraspinal muscles (96.33 $\pm$ 2.31 SD).



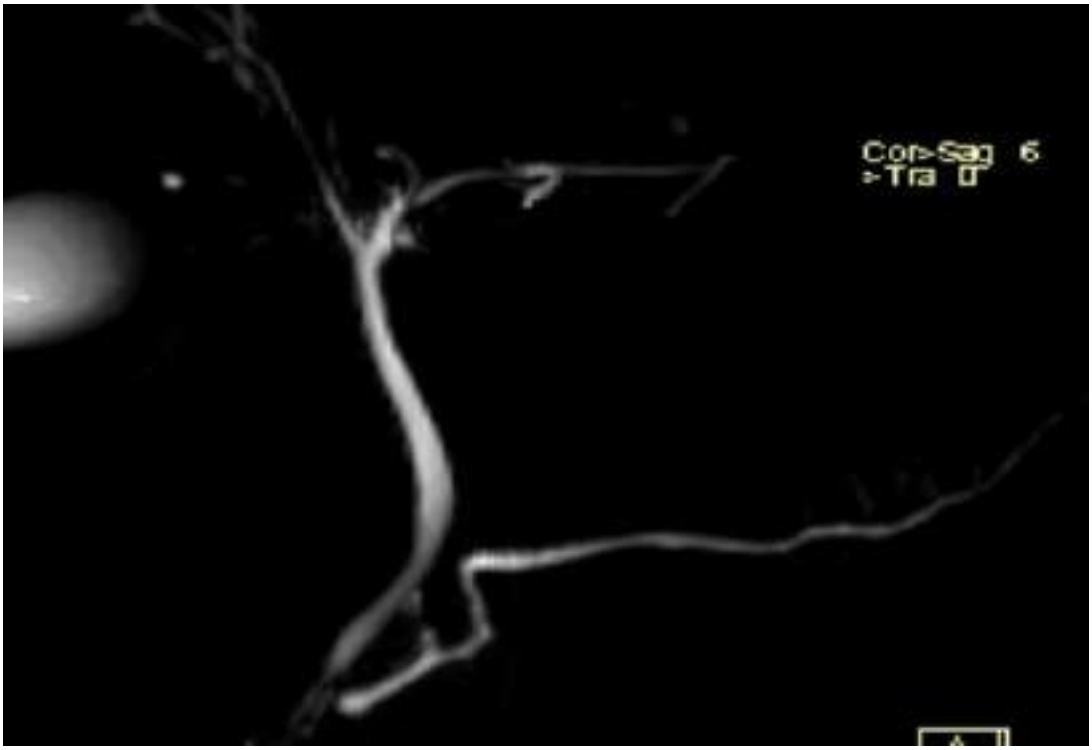
Picture 13) (No; 75 as in the masterchart) MIP image in the same patient revealed the normal ductal morphology.





Picture 14, 15) (No; 49 as in the masterchart) A patient was asymptomatic and underwent MRCP. This is T1 weighted fat sat image which showed the mean signal intensity of pancreas by placing ROI i.e. (272.50 $\pm$ 21.08 SD), Liver (377.66 $\pm$ 7.37 SD), Spleen (342.00 $\pm$ 9.7 SD), Paraspinal muscles (293.00 $\pm$ 8.60 SD).

As the patient was asymptomatic but the signal intensity of the pancreas was lower as compared to liver, spleen & paraspinal muscles.



Picture 16) (No; 49 as in the masterchart) MIP image in the above patient revealed normal ductal morphology

KEY TO MASTER CHART

M	MALE
F	FEMALE
CRP	C- REACTIVE PROTEIN
PD	PANCREATIC DUCT
Y	YES
N	NO
SI	SIGNAL INTENSITY
P	PANCREAS
PSM	PARASPINAL MUSLES
L	LIVER
S	SPLEEN
SIR	SIGNAL INTENSITY RATIO

S. No.	Name	MRD	Age	Age group	Sex	Clinical features	Elastase (mcg/g)	S.Amylase	S.Lipase	CRP	MPD Calibre (mm)	Location	Ductal changes	No; of PD side branches	PD Contour	Severity of ductal changes	Intraductal stricture	Intraductal Calculus	Only signal changes
1	RK	881610	27	1	M	Epigastric pain	150	34	50	10	4	Body	Yes	>3	Irregular	Grade 4-Severe	Present	Absent	No
2	HS	846873	13	1	M	Weight loss	133	28	43	8	3	Head	Yes	>3	Irregular	Grade 4-Severe	Present	Absent	No
3	IK	913328	47	3	F	Weight loss	not done	30	45	20	8	Head	Yes	multiple	Irregular	Grade 3-Moderate	Absent	Absent	No
4	GS	706786	52	3	M	Epigastric pain	182	29	42	17	3.8	Head	Yes	absent	Irregular	Grade 4-Severe	Present	Absent	No
5	KK	935366	30	2	F	Weight loss	167	31	50	12	1.5	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Yes
6	RS	296723	22	1	M	Weight loss	174	40	54	9	4.6	Body	Yes	>3	Irregular	Grade 4-Severe	Present	Present	No
7	HY	528866	27	1	M	Steatorrhea	158	33	43	13	7.2	Body	Yes	>3	Irregular	Grade 4-Severe	Present	Absent	No
8	GT	686657	26	1	M	Epigastric pain	177	46	95	12	2.7	Head	Yes	>3	Irregular	Grade 3-Moderate	Absent	Absent	Yes
9	GR	1086983	22	1	M	Steatorrhea	156	38	50	16	2.5	Body	Yes	multiple	Irregular	Grade 3-Moderate	Absent	Absent	No
10	RD	492108	13	1	F	Weight loss	134	39	86	20	2	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Yes
11	DK	1033524	15	1	F	Weight loss	182	42	53	18	2	Body	Yes	>3	Irregular	Grade 3-Moderate	Absent	Absent	No
12	HK	973636	28	1	M	Epigastric pain	167	41	40	13	1.9	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Yes
13	SS	1005625	31	2	M	Steatorrhea	152	28	69	9	13	Body	Yes	<3	Irregular	Grade 4-Severe	Present	Present	No
14	DS	37308	43	3	M	Epigastric pain	145	25	74	11	4.7	Head	Yes	>3	Irregular	Grade 4-Severe	Present	Absent	No
15	GV	833797	27	1	M	Epigastric pain	126	32	54	16	3.5	Body	Yes	>3	Regular	Grade 3-Moderate	Absent	Absent	No
16	GU	907307	40	2	M	Steatorrhea	137	34	62	17	6.5	Body	Yes	few	Irregular	Grade 3-Moderate	Absent	Absent	No
17	AK	1008220	43	3	M	Weight loss	148	40	43	16	2	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Yes
18	SE	921887	37	2	M	Steatorrhea	162	38	55	17	12	Body	Yes	>3	Irregular	Grade 4-Severe	Absent	Present	No
19	FT	944091	37	2	M	Epigastric pain	187	33	53	12	4	Body	Yes	>3	Irregular	Grade 4-Severe	Present	Absent	No
20	FR	1053992	45	3	M	Epigastric pain	not done	32	63	14	7.5	Head	Yes	>3	Irregular	Grade 4-Severe	Present	Absent	No
21	UG	985023	25	1	M	Weight loss	162	28	63	16	1.5	Body	Yes	absent	Irregular	Grade 4-Severe	Present	Absent	No
22	FV	972981	36	2	M	Epigastric pain	143	32	54	10	5	Body	No	>3	Irregular	Grade 0 - normal	Absent	Absent	Yes
23	LS	578876	36	2	M	Weight loss	182	45	45	16	9	Head	Yes	multiple	Irregular	Grade 4-Severe	Present	Present	No
24	JV	930419	30	2	M	Weight loss	156	41	54	19	1.9	Body	Yes	multiple	Regular	Grade 4-Severe	Present	Absent	No
25	KY	96833	29	1	M	Epigastric pain	176	35	67	11	4	Head	Yes	absent	Irregular	Grade 3-Moderate	Absent	Absent	No
26	GT	70819	10	1	F	Epigastric pain	145	27	52	17	3	Body	Yes	>3	Irregular	Grade 4-Severe	Present	Absent	No
27	DV	1046543	34	2	M	Weight loss	not done	32	43	16	5	Body	Yes	multiple	Irregular	Grade 4-Severe	Present	Absent	No
28	VV	758122	16	1	M	Epigastric pain	117	42	54	13	2.6	Body	Yes	absent	Regular	Grade 4-Severe	Present	Absent	No
29	SR	907224	40	2	M	Weight loss	176	38	63	8	4	Body	Yes	absent	Regular	Grade 4-Severe	Present	Absent	No
30	AK	504051	21	1	M	Steatorrhea	125	26	54	10	5	Body	Yes	multiple	Irregular	Grade 4-Severe	Present	Absent	No
31	LK	1031082	40	2	F	Epigastric pain	153	25	69	18	3	Body	Yes	absent	Irregular	Grade 4-Severe	Present	Absent	No
32	HV	1121868	27	1	M	Steatorrhea	165	34	45	13	3.5	Body	Yes	multiple	Irregular	Grade 3-Moderate	Absent	Absent	No
33	MS	144757	30	2	M	Weight loss	130	26	65	11	4	Body	Yes	multiple	Irregular	Grade 4-Severe	Present	Absent	No
34	MM	356202	34	2	M	Epigastric pain	186	37	54	15	1.9	Head	No	absent	Regular	Grade 0 - normal	Absent	Absent	Yes
35	SA	518720	23	1	M	Steatorrhea	179	27	75	12	2	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Yes
36	FD	871293	19	1	M	Weight loss	not done	26	74	21	6	Body	Yes	>3	Irregular	Grade 4-Severe	Absent	Present	No
37	HG	879271	65	3	F	Epigastric pain	154	32	68	17	2.9	Body	Yes	>3	Irregular	Grade 3-Moderate	Absent	Absent	No
38	BZ	739053	55	3	M	Steatorrhea	not done	46	64	18	14	Head	Yes	>3	Tortous	Grade 4-Severe	Absent	Present	No
39	ME	412900	58	3	M	Weight loss	145	49	74	20	5	Head	Yes	>3	Tortous	Grade 3-Moderate	Absent	Absent	No
40	KS	97546	26	1	M	Epigastric pain	132	37	67	16	8	Head	Yes	>3	Irregular	Grade 4-Severe	Present	Present	No
41	YF	1051634	24	1	M	steatorrhea	117	38	75	18	13	Head	Yes	>3	Irregular	Grade 4-Severe	Present	Present	No
42	DD	74031	52	3	M	Weight loss	157	27	60	18	4	Body	Yes	absent	Irregular	Grade 4-Severe	Present	Absent	No
43	EG	1024974	40	2	M	Weight loss	134	30	54	21	7	Body	Yes	multiple	Irregular	Grade 4-Severe	Present	Absent	No
44	IS	238345	35	2	M	Epigastric pain	not done	36	36	15	11	Head	Yes	multiple	Irregular	Grade 4-Severe	Present	Absent	No
45	EG	1032613	25	1	M	Weight loss	172	32	45	19	13	Body	YES	multiple	Irregular	Grade 4-Severe	Present	Present	No
46	JS	491749	39	2	F					5	1.5	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
47	ML	1134805	30	2	M					3	1.7	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
48	BF	1007398	40	2	M					7	1.5	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	yes
49	MC	1128380	28	1	M					11	1.6	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Yes
50	VL	695246	37	2	M					14	1.5	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	yes

S. No.	Name	MRD	SI + ductal changes	Signal	Ductal		Parenchymal T1 SI(Pancreas)	T1 SI (Liver)	T1 SI (Spleen)	T1 SI PSM	SIR(P/L)-1	SIR(P/S)-1	SIR(P/PSM)-1	Parenchymal Thickness		Distribution	Follow Up
1	RK	881610	Signal+Ductal	Y	Y	2	278	345	378	321	-0.19	-0.26	-0.13	17	17 mm in body	50%	Medical T/t
2	HS	846873	Signal+Ductal	Y	Y	2	134	141	109	103	-0.05	0.23	0.30	23	23mm in body	<30%	Medical T/t
3	IK	913328	Signal+Ductal	Y	Y	2	170	138	264	214	0.23	-0.36	-0.21	15	1.5 cm in B	50%	Medical T/t
4	GS	706786	Signal+Ductal	Y	Y	2	172	230	164	171	-0.25	0.05	0.01	20	20mm in body	50%	Medical T/t
5	KK	935366	Signal	Y	N	1	98	122	129	124	-0.20	-0.24	-0.21	20	20 mm in body	<30%	Medical T/t
6	RS	296723	Signal+Ductal	Y	Y	2	227	311	251	232	-0.27	-0.10	-0.02	19	19mm in body	70%	Medical T/t
7	HY	528866	Signal+Ductal	Y	Y	2	195	296	244	212	-0.34	-0.20	-0.08	15	15mm in body	50%	Medical T/t
8	GT	686657	Signal	Y	N	1	113	88	82	80	0.28	0.38	0.41	14	14mm in body	30%	Medical T/t
9	GR	1086983	Signal+Ductal	Y	Y	2	180	266	288	256	-0.32	-0.38	-0.30	6	6mm in neck&body	70%	Medical T/t
10	RD	492108	Signal	Y	N	1	48	93	70	64	-0.48	-0.31	-0.25	23	2.3 cm in body	30%	Medical T/t
11	DK	1033524	Signal+Ductal	Y	Y	2	280	284	249	245	-0.01	0.12	0.14	15	15 mm in head	50%	Medical T/t
12	HK	973636	Signal	Y	N	1	90	142	122	112	-0.37	-0.26	-0.20	10	10 mm in body	50%	Medical T/t
13	SS	1005625	Signal+Ductal	Y	Y	2	171	341	225	190	-0.50	-0.24	-0.10	6	6 mm in Body	70%	Medical T/t
14	DS	37308	Signal+Ductal	Y	Y	2	75	133	113	95	-0.44	-0.34	-0.21	8	19mm H,10mmB,8mmT	70%	Medical T/t
15	GV	833797	Signal+Ductal	Y	Y	2	331	337	254	232	-0.02	0.30	0.43	12	12 mm in head	50%	Medical T/t
16	GU	907307	Signal+Ductal	Y	Y	2	181	292	184	197	-0.38	-0.02	-0.08	20	20 mm in body	50%	Medical T/t
17	AK	1008220	Signal	Y	N	1	58	98	67	59	-0.41	-0.13	-0.02	15	15-16 mm in prox body	50%	Medical T/t
18	SE	921887	Signal+Ductal	Y	Y	2	172	233	158	184	-0.26	0.09	-0.07	6	6-7 mm in body	70%	Medical T/t
19	FT	944091	Signal+Ductal	Y	Y	2	82	88	78	72	-0.07	0.05	0.14	12	12mmH,15mm in B	70%	Medical T/t
20	FR	1053992	Signal+Ductal	Y	Y	2	187	272	225	200	-0.31	-0.17	-0.06	10	1.6cmH,1cm B,1.4cm T	70%	Medical T/t
21	UG	985023	Signal+Ductal	Y	Y	2	186	285	217	225	-0.35	-0.14	-0.17	9	1.8cm H,0.9mmB,1.4mmT	50%	Medical T/t
22	FV	972981	Signal	Y	N	1	66	95	92	66	-0.31	-0.28	0.00	10	10 mm indistal body	50%	Medical T/t
23	LS	578876	Signal+Ductal	Y	Y	2	122	256	178	169	-0.52	-0.31	-0.28	12	1.2 cm in B& T	>70%	Medical T/t
24	JV	930419	Signal+Ductal	Y	Y	2	280	298	295	285	-0.06	-0.05	-0.02	16	16mm in body	50%	Medical T/t
25	KY	96833	Signal+Ductal	Y	Y	2	96	130	99	112	-0.26	-0.03	-0.14	10	10 mm in body	70%	Medical T/t
26	GT	70819	Signal+Ductal	Y	Y	2	297	339	318	300	-0.12	-0.07	-0.01	12	12-13mm in body	50%	Medical T/t
27	DV	1046543	Signal+Ductal	Y	Y	2	292	396	301	298	-0.26	-0.03	-0.02	16	16 mm in body	50%	Medical T/t
28	VV	758122	Signal+Ductal	Y	Y	2	386	414	251	290	-0.07	0.54	0.33	23	2.3 cm in tail	50%	Medical T/t
29	SR	907224	Signal+Ductal	Y	Y	2	324	348	268	258	-0.07	0.21	0.26	10	1 cm in Body & tail	50%	Medical T/t
30	AK	504051	Signal+Ductal	Y	Y	2	208	286	294	255	-0.27	-0.29	-0.18	10	10 mm in body	70%	Medical T/t
31	LK	1031082	Signal+Ductal	Y	Y	2	342	341	319	255	0.00	0.07	0.34	20	2cm in B	30%	Medical T/t
32	HV	1121868	Signal+Ductal	Y	Y	2	165	201	220	208	-0.18	-0.25	-0.21	15	15 mm in body	30%	Medical T/t
33	MS	144757	Signal+Ductal	Y	Y	2	260	260	305	255	0.00	-0.15	0.02	26	2.6 cm in body	50%	Medical T/t
34	MM	356202	Signal	Y	N	1	162	322	259	233	-0.50	-0.37	-0.30	20	20 mm in body	30%	Medical T/t
35	SA	518720	Signal	Y	N	2	272	347	309	258	-0.22	-0.12	0.05	5	5mm in prox body	30%	Medical T/t
36	FD	871293	Signal+Ductal	Y	Y	2	271	344	268	257	-0.21	0.01	0.05	16	16 mm	>70%	PD stenting
37	HG	879271	Signal+Ductal	Y	Y	2	228	330	306	244	-0.31	-0.25	-0.07	20	2cm in B	30%	PD stenting
38	BZ	739053	Signal+Ductal	Y	Y	2	170	284	243	191	-0.40	-0.30	-0.11	10	1cm in body	>70%	PD stenting
39	ME	412900	Ductal	N	Y	2	365	300	289	255	0.22	0.26	0.43	s	1.2cm H,1.1 cm B,1cm T	<30%	PD stenting
40	KS	97546	Signal+Ductal	Y	Y	2	55	74	61	54	-0.26	-0.10	0.02	14	14mmB,25 mm T	70%	PD stenting
41	YF	1051634	Ductal	N	Y	2	268	268	211	187	0.00	0.27	0.43	6	6-7 mm in tail	>70%	PD stenting
42	DD	74031	Ductal	N	Y	2	256	256	244	230	0.00	0.05	0.11	10	14mm in B,10mm in tail	50%	PD stenting
43	EG	1024974	Ductal	N	Y	2	298	298	275	224	0.00	0.08	0.33	6	6-7 mm in body	50%	PD stenting
44	IS	238345	Ductal	N	Y	2	290	290	287	241	0.00	0.01	0.20	8	8 mm in H, 9 mm in Tail	70%	PD stenting
45	EG	1032613	Ductal	N	Y	2	255	255	224	212	0.00	0.14	0.20	10	1 cm in Body & tail	70%	PD stenting
46	JS	491749	Absent	N	N	0	340	293	263	220	0.16	0.29	0.55	26	26 mm in body		
47	ML	1134805	Absent	N	N	0	308	287	226	192	0.07	0.36	0.60	25	25 mm in body		
48	BF	1007398	Signal	Y	N	0	272	377	342	293	-0.28	-0.20	-0.07	24	24 mm in body		
49	MC	1128380	Signal	Y	N	0	297	325	313	305	-0.09	-0.05	-0.03	25	25 mm in body		
50	VL	695246	Signal	Y	N	0	320	370	346	330	-0.14	-0.08	-0.03	23	23mm in body		

S. No.	Name	MRD
1	RK	881610
2	HS	846873
3	IK	913328
4	GS	706786
5	KK	935366
6	RS	296723
7	HY	528866
8	GT	686657
9	GR	1086983
10	RD	492108
11	DK	1033524
12	HK	973636
13	SS	1005625
14	DS	37308
15	GV	833797
16	GU	907307
17	AK	1008220
18	SE	921887
19	FT	944091
20	FR	1053992
21	UG	985023
22	FV	972981
23	LS	578876
24	JV	930419
25	KY	96833
26	GT	70819
27	DV	1046543
28	VV	758122
29	SR	907224
30	AK	504051
31	LK	1031082
32	HV	1121868
33	MS	144757
34	MM	356202
35	SA	518720
36	FD	871293
37	HG	879271
38	BZ	739053
39	ME	412900
40	KS	97546
41	YF	1051634
42	DD	74031
43	EG	1024974
44	IS	238345
45	EG	1032613
46	JS	491749
47	ML	1134805
48	BF	1007398
49	MC	1128380
50	VL	695246

S. No.	Name	MRD	Age	Age group	Sex	Clinical features	Elastase (mcg/g)	S.Amylase	S.Lipase	CRP	MPD Calibre (mm)	Location	Ductal changes	No; of PD side branches	PD Contour	Severity of ductal changes	Intraductal stricture	Intraductal Calculus	Only signal changes
51	AA	1149560	34	2	F					5	1.4	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Yes
52	YS	1150571	45	3	M					7	1.5	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Yes
53	PS	1129536	27	1	M					6	1.6	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Yes
54	SS	866557	42	3	M					10	1.7	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Yes
55	MSS	1017745	38	2	M					2	1.9	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
56	JSB	1024012	43	3	F					4	2	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
57	MD	1088720	26	1	M					5	1.8	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
58	FA	1076373	35	2	M					2	1.6	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
59	YR	1167089	37	2	M					6	1.7	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
60	IG	1032245	36	2	M					2	1.5	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
61	SM	1025415	29	1	M					5	1.6	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
62	MK	992954	36	2	M					4	1.7	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
63	LS	1025483	45	3	M					2	1.6	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
64	DE	1022182	40	2	M					5	1.5	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
65	SG	1020093	37	2	F					3	1.6	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
66	JD	1010813	24	1	M					2	1.7	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
67	VS	1150631	39	2	M					1	1.8	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
68	LO	1011800	40	2	M					4	1.7	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
69	JH	529232	29	1	M					2	1.6	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
70	YG	1012421	47	3	F					5	1.6	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
71	UH	1012548	35	2	M					2	1.7	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
72	EF	466358	24	1	M					4	1.5	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
73	BS	1156918	17	1	F					5	1.6	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
74	MG	1050886	45	3	M					2	1.7	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
75	YY	1134044	42	3	M					1	1.6	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
76	YG	1142548	43	3	M					4	1.5	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
77	EC	635500	46	3	F					2	1.7	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
78	BB	1006821	25	1	F					3	1.8	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
79	DMS	1137140	58	3	M					3	1.6	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
80	CS	1007922	26	1	M					4	1.7	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
81	ZF	1068912	23	1	M					2	1.4	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
82	SL	1146573	43	3	M					4	1.6	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
83	MJ	1015615	23	1	M					3	1.8	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
84	JD	1128993	40	2	M					2	1.6	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
85	GSJ	1132385	25	1	M					4	1.7	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
86	DEW	278892	39	2	M					3	1.5	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
87	VSA	1155676	29	1	M					2	1.9	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
88	BSZ	1154752	30	2	M					3	1.6	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
89	MML	1167536	34	2	M					2	1.5	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal
90	IS	635500	49	3	M					3	1.7	Body	No	absent	Regular	Grade 0 - normal	Absent	Absent	Normal

S. No.	Name	MRD	SI + ductal changes	Signal	Ductal		Parenchymal T1 SI(Pancreas)	T1 SI (Liver)	T1 SI (Spleen)	T1 SI PSM	SIR(P/L)-1	SIR(P/S)-1	SIR(P/PSM)-1	Parenchymal Thickness		Distribution	Follow Up
51	AA	1149560	Signal	Y	N	0	380	426	387	384	-0.11	-0.02	-0.01	24	24 mm in body		
52	YS	1150571	Signal	Y	N	0	310	330	319	312	-0.06	-0.03	-0.01	23	23 mm in body		
53	PS	1129536	Signal	Y	N	0	352	380	365	359	-0.07	-0.04	-0.02	24	24 mm in body		
54	SS	866557	Signal	Y	N	0	282	320	300	295	-0.12	-0.06	-0.04	23	23 mm in body		
55	MSS	1017745	Absent	N	N	0	311	282	255	223	0.10	0.22	0.39	21	21mm in body		
56	JSB	1024012	Absent	N	N	0	319	278	255	221	0.15	0.25	0.44	22	22 mm in body		
57	MD	1088720	Absent	N	N	0	411	358	330	220	0.15	0.25	0.87	23	23 mm in body		
58	FA	1076373	Absent	N	N	0	336	235	214	205	0.43	0.57	0.64	21	21 mm in body		
59	YR	1167089	Absent	N	N	0	272	230	212	216	0.18	0.28	0.26	22	22 mm in body		
60	IG	1032245	Absent	N	N	0	311	267	254	221	0.16	0.22	0.41	23	23mm in body		
61	SM	1025415	Absent	N	N	0	355	325	274	221	0.09	0.30	0.61	22	22 mm in body		
62	MK	992954	Absent	N	N	0	352	322	288	220	0.09	0.22	0.60	23	23 mm in body		
63	LS	1025483	Absent	N	N	0	311	271	247	220	0.15	0.26	0.41	22	22 mm in body		
64	DE	1022182	Absent	N	N	0	426	373	302	225	0.14	0.41	0.89	23	23 mm in body		
65	SG	1020093	Absent	N	N	0	313	271	252	220	0.15	0.24	0.42	22	22 mm in body		
66	JD	1010813	Absent	N	N	0	316	278	255	230	0.14	0.24	0.37	23	23mm in body		
67	VS	1150631	Absent	N	N	0	321	279	253	230	0.15	0.27	0.40	21	21 mm in body		
68	LO	1011800	Absent	N	N	0	321	280	253	232	0.15	0.27	0.38	22	22 mm in body		
69	JH	529232	Absent	N	N	0	355	228	225	202	0.56	0.58	0.76	23	23 mm in body		
70	YG	1012421	Absent	N	N	0	314	282	258	215	0.11	0.22	0.46	22	22 mm in body		
71	UH	1012548	Absent	N	N	0	325	312	255	230	0.04	0.27	0.41	23	23 mm in body		
72	EF	466358	Absent	N	N	0	341	321	262	201	0.06	0.30	0.70	21	21 mm in body		
73	BS	1156918	Absent	N	N	0	401	310	316	198	0.29	0.27	1.03	21	21mm in body		
74	MG	1050886	Absent	N	N	0	158	108	128	96	0.46	0.23	0.65	23	23 mm in body		
75	YY	1134044	Absent	N	N	0	322	241	233	212	0.34	0.38	0.52	22	22 mm in body		
76	YG	1142548	Absent	N	N	0	336	234	271	212	0.44	0.24	0.58	23	23 mm in body		
77	EC	635500	Absent	N	N	0	323	279	249	230	0.16	0.30	0.40	21	21 mm in body		
78	BB	1006821	Absent	N	N	0	433	326	323	220	0.33	0.34	0.97	22	22 mm in body		
79	DMS	1137140	Absent	N	N	0	318	242	218	201	0.31	0.46	0.58	21	21 mm in body		
80	CS	1007922	Absent	N	N	0	415	331	329	221	0.25	0.26	0.88	23	23 mm in body		
81	ZF	1068912	Absent	N	N	0	385	305	272	202	0.26	0.42	0.91	22	22 mm in body		
82	SL	1146573	Absent	N	N	0	336	232	250	232	0.45	0.34	0.45	21	21 mm in body		
83	MJ	1015615	Absent	N	N	0	408	360	323	225	0.13	0.26	0.81	23	23 mm in body		
84	JD	1128993	Absent	N	N	0	324	221	221	212	0.47	0.47	0.53	21	21 mm in body		
85	GSJ	1132385	Absent	N	N	0	340	247	220	215	0.38	0.55	0.58	21	21 mm in body		
86	DEW	278892	Absent	N	N	0	320	271	251	230	0.18	0.27	0.39	22	22 mm in body		
87	VSA	1155676	Absent	N	N	0	432	333	328	205	0.30	0.32	1.11	21	21mm in body		
88	BSZ	1154752	Absent	N	N	0	338	221	213	200	0.53	0.59	0.69	22	22 mm in body		
89	MML	1167536	Absent	N	N	0	422	324	310	208	0.30	0.36	1.03	22	22 mm in body		
90	IS	635500	Absent	N	N	0	323	279	249	230	0.16	0.30	0.40	21	21 mm in body		

S. No.	Name	MRD
51	AA	1149560
52	YS	1150571
53	PS	1129536
54	SS	866557
55	MSS	1017745
56	JSB	1024012
57	MD	1088720
58	FA	1076373
59	YR	1167089
60	IG	1032245
61	SM	1025415
62	MK	992954
63	LS	1025483
64	DE	1022182
65	SG	1020093
66	JD	1010813
67	VS	1150631
68	LO	1011800
69	JH	529232
70	YG	1012421
71	UH	1012548
72	EF	466358
73	BS	1156918
74	MG	1050886
75	YY	1134044
76	YG	1142548
77	EC	635500
78	BB	1006821
79	DMS	1137140
80	CS	1007922
81	ZF	1068912
82	SL	1146573
83	MJ	1015615
84	JD	1128993
85	GSJ	1132385
86	DEW	278892
87	VSA	1155676
88	BSZ	1154752
89	MML	1167536
90	IS	635500

Baba Farid University
of Health Sciences
Sadiq Road
Faridkot-151 203 (Pb). INDIA.
Phone : 01639-256232,256236
Tele/Fax : 01639-256235
Email – Examination@bfuhs.ac.in

45

No. BFUHS/2K21p-TH/ 5497 Date 2/4/2021

The Principal,
DMC,Ludhiana.

Subject: **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 22.01.2021 has approved the plan of thesis of **Dr. Gagandeep Kaur**, a student of your college for **MD Radio-Diagnosis** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis
Role of magnetic resonance imaging in chronic pancreatitis

Supervisor
Dr. Chandan Kakkar
Assoc. Prof.,
Dept. of Radio-Diagnosis,
DMCH, Ludhiana.

Co-Supervisor
Dr. Ritu Dhawan Galhotra
Prof.,
Dept. of Radio-Diagnosis,
DMCH, Ludhiana.

Dr. Ramit Mahajan
Asst. Prof.,
Dept. of Gastroenterology,

The student has to submit the three copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

S P Singh

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

1. ***If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.***
3. *The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.*

ROLE OF MAGNETIC RESONANCE IMAGING IN CHRONIC PANCREATITIS

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

MD

In

RADIODIAGNOSIS

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

November, 2019

Dr. Gagandeep Kaur

DEPARTMENT OF RADIODIAGNOSIS
DAYANAND MEDICAL COLLEGE AND HOSPITAL
LUDHIANA

ABSTRACT OF PLAN OF THESIS

Title	ROLE OF MAGNETIC RESONANCE IMAGING IN CHRONIC PANCREATITIS
For the degree of	Radiodiagnosis
Name of the candidate	Dr. Gagandeep Kaur
Supervisor	Dr. Chandan Kakkar, Associate Professor, Department of Radiodiagnosis, DMCH
Co-supervisors	Dr. Ritu Dhawan Galhotra, Associate Professor, Department of Radiodiagnosis, DMCH Dr. Ramit Mahajan, Assistant Professor, Department of Gastroenterology, DMCH
Institution	Dayanand Medical College & Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: Chronic pancreatitis is characterized by continued inflammation and destruction that leads to irreversible morphological changes in the pancreatic parenchyma & its ductal anatomy. It is widely acknowledged that MRI with MR cholangiopancreatography is more sensitive than is CT for detection of ductal and subtle parenchymal changes, especially during early stages of chronic pancreatitis. MR cholangiopancreatography thus provides information on both ductal and parenchymal features of chronic pancreatitis such as T1 signal changes, focal or diffuse gland atrophy and irregular contour of the pancreatic duct.

Aim of study: 1) To study the morphological, ductal and signal intensity pattern changes of pancreas in patients with chronic pancreatitis.

Materials and methods: All patients who are diagnosed or suspected to have chronic pancreatitis and are referred for MRI abdomen to the Department of Radiodiagnosis, DMCH Ludhiana for a period of one and half year from 1st Dec 2019 to 31st May 2021.

The diagnosed patients will be assessed for morphological changes in pancreas with emphasis on signal intensity pattern and ductal changes in chronic pancreatitis. The signal intensity pattern of the patients with chronic pancreatitis will be compared with age and sex matched controls to assess and define the range of values in patients and control population.

Inclusion criteria 1) Clinically diagnosed patients of chronic pancreatitis.

2) Patients with biochemical, alternative imaging and other clinical parameters suggestive of chronic pancreatitis.

Exclusion criteria: 1) If the patient refuses to participate in the study

2) Contraindications to MRI as per the checklist on the departmental consent form 3) Acute pancreatitis, 4) Known cystic or solid pancreatic neoplasm 5) Incomplete or suboptimal quality of MRI examinations

Clinical significance: The clinical significance of MRI in cases of chronic pancreatitis lies in the role it plays in deciding the adequate treatment protocol like maintaining the lost function of pancreas pharmacologically. It helps to determine the treatment, artificially maintain the endocrine & exocrine function of pancreas as well as maintaining serum calcium levels.

Keywords: Chronic pancreatitis---MRI---Pancreatic duct changes---Pancreatic signal intensity changes---most sensitive & specific parameter.

(Signature) Candidate	(Signature) Co-supervisor	(Signature) Co-supervisor	(Signature) Supervisor	(Signature) Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"ROLE OF MAGNETIC RESONANCE IMAGING IN CHRONIC PANCREATITIS"** exist in the Department of Radiodiagnosis, DMC&H, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis is genuine and is collected by the candidate herself under our supervision and guidance.

Supervisor

Dr. Chandan Kakkar
Associate Professor
Department of Radiodiagnosis
DMCH, Ludhiana

Co-Supervisor(s)

Dr. Ritu D Galhotra
Associate Professor
Department of Radiodiagnosis
DMCH, Ludhiana

Dr. Ramit Mahajan
Assistant Professor
Department of Gastroenterology
DMCH, Ludhiana

HEAD OF DEPARTMENT

Dr. Kavita Saggar
Professor and Head
Department of Radiodiagnosis
DMCH, Ludhiana

Place: _____
Date: _____

APPROVAL PROFORMA
BY
RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

Name of candidate	Dr. Gagandeep Kaur
Department	Radiodiagnosis
Topic of Thesis	ROLE OF MAGNETIC RESONANCE IMAGING IN CHRONIC PANCREATITIS
Likely date of appearing in PG examination	May-June 2022
Date of enrollment	1st May 2019
Name of Head of Department	Dr. Kavita Saggar
1) Supervisor **2) Co-Supervisor(s)**	Dr. Chandan Kakkar Dr. Ritu D Galhotra Dr. Ramit Mahajan
Signature of Members of Research Committee with Stamp	**Signature of Members of Ethics Committee with Stamp**
Dr. Jaspal Singh (Professor & Head, Surgery)	Mr. A K Jindal (Member, Ethics Committee)
Dr. Dinesh Gupta (Professor & Head, Medicine)	Dr. Anu Sharma (Professor, Anatomy)
Dr. Sandeep Kaushal (Assistant Dean Academics)	Dr. Deepinder K Chhina (Professor & Head, Microbiology)
Dr. Rajoo Singh Chhina (Dean Academics)	Dr. Neena Sood (Professor & Head, Pathology)
Dr. Gurpreet S Wander (Vice Principal)	Dr. Gurpreet S Wander (Vice Principal)
	Dr. Gagandeep Singh (Secretary, Ethics Committee Professor & Head, Neurology)

Approved: Yes / No

Dr. Sandeep Puri
Chairperson
Research Committee

Chairperson
Research Committee

Approved: Yes / No

Prof. Arvind Malhotra
Chairperson
Ethics Committee

Chairperson
Ethics Committee

BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT

PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

1	NAME OF THE CANDIDATE AND ADDRESS	Dr Gagandeep Kaur H/No 285/3h Golden Bagh Jagraon (Ludhiana).
2	NAME OF THE INSTITUTE	Dayanand Medical College and Hospital, Ludhiana
3	COURSE OF STUDY AND SUBJECT	M.D. Radiodiagnosis
4	DATE OF ADMISSION OF COURSE	01/05/2019
5	TITLE OF THE TOPIC	ROLE OF MAGNETIC RESONANCE IMAGING IN CHRONIC PANCREATITIS

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

MRI of the pancreas has undergone a major change due to recent technical improvements, including the capability of providing high resolution images of the pancreatic duct with the use of half-Fourier T2-weighted pulse sequences and angiographic depiction of blood vessels with three-dimensional MR angiography sequences. The potential advantage of MRI relative to multidetector CT is its ability to show altered signal intensity in pancreatic disease without the need for contrast material injection. The normal pancreas has been reported to be hyperintense relative to all other solid abdominal tissues on T1-weighted images in multiple studies.[1] Chronic pancreatitis has been defined as a continuing inflammatory disease of the pancreas characterized by irreversible morphological changes that typically cause pain and/or permanent loss of function.[2] The diagnosis of chronic pancreatitis can be made by histologic or morphologic criteria alone or by a combination of morphologic, functional and clinical findings.[3]

MRI and MRCP play an important role in the diagnosis of chronic pancreatitis (CP) by imaging pancreatic parenchyma and ducts.[4] MRI/MRCP is more widely used than computed tomography (CT) for mild to moderate CP due to its increased sensitivity for pancreatic ductal and gland changes; however, it does not detect the calcifications seen in advanced CP. Quantitative MR imaging offers potential advantages over conventional qualitative imaging, including simplicity of analysis, quantitative and population-based comparisons, and more direct interpretation of detected changes. These techniques may provide quantitative metrics for determining the presence and severity of acinar cell loss and aid in the diagnosis of chronic pancreatitis. Given the fact that the parenchymal changes of CP precede the ductal involvement, there would be a significant benefit from developing MRI/MRCP-based, more robust diagnostic criteria combining ductal and parenchymal findings. Role of MRI/MRCP in the diagnosis of CP has been acknowledged by recent guidelines of the American

Pancreatic Association and a modified Cambridge classification for MR/MRCP and CT has been proposed.[5]

Among cross sectional imaging modalities, multi-detector CT (MDCT) has been a cornerstone for evaluating chronic pancreatitis (CP)since it is ubiquitous, assesses primary disease process, identifies complications like pseudocyst or vascular thrombosis with high sensitivity and specificity, guides therapeutic management decisions, and provides images with isotropic resolution within seconds. Conventional MDCT has certain limitations and is reserved to provide predominantly morphological (e.g., calcifications, organ size) rather than functional information. The emerging applications of radiomics and artificial intelligence are poised to extend the current capabilities of MDCT.

Conventional grayscale B-mode US is often the first imaging performed for abdominal assessment. Transabdominal US (TA-US) however may be limited for the pancreas due to retroperitoneal location of the gland. To overcome this, detailed pancreatic evaluation is generally performed with endoscopic ultrasound (EUS). Advances such as harmonic imaging, US elastography and contrast-enhanced US (CE-US) can be applied to both abdominal and endoscopic approaches. Tissue harmonic US imaging improves the resolution with reduced reverberation artifacts.[6]

Conventional transabdominal B-mode US has a lower accuracy than MR in determining fatty infiltration and consequently estimating pancreatic exocrine function.[7] US elastography can be used as a quantitative measure to estimate pancreatic stiffness using either strain or shear wave techniques. Despite these advances, the retroperitoneal location of pancreas and invasive nature of EUS makes CT and MR the modality of choice for imaging.

The use of MRI to perform magnetic resonance cholangiopancreatography (MRCP) is evolving ,as an important tool in the evaluation of chronic pancreatitis.[8] MRCP is noninvasive, avoids ionizing radiation and contrast administration, and does not routinely require sedation, making it a diagnostic procedure of shot, fast spin

echo sequences during 2-second suspended respirations. Although studies have begun to demonstrate the important role MRI and MRCP can play in the diagnosis and staging of pancreatic cancer, investigation into their role in chronic pancreatitis has only recently begun. Major lesions such as grossly dilated ducts, communicating pseudocysts and even pancreas divisum can be detected.

6.2 REVIEW OF LITERATURE

A theoretical advantage of MR in the diagnosis of pancreatic disease is the high contrast resolution between different tissues it makes possible and its ability to show altered signal intensity even in the absence of gross morphological abnormality.[9] Chronic pancreatitis (CP) involves progressive inflammatory changes in the pancreas characterized by chronic unrelenting pain with episodic flares, impairment of exocrine and endocrine function. The diagnosis of CP relies on relevant symptoms and diagnostic modalities to assess pancreatic structure and function. An important factor for imaging of CP is the presence of fatty infiltration and parenchymal atrophy, which are two commonly observed manifestations of the disease.

Soto JA did a study to determine the role of three-dimensional fast spin echo magnetic resonance (MR) cholangiopancreatography in the evaluation of the normal and abnormal pancreatic duct. A non-breath-hold MR cholangiopancreatographic technique with use of a body coil was compared with direct pancreatography performed with endoscopic retrograde cholangiopancreatography in 37 patients. They concluded that MR cholangiopancreatography can accurately demonstrate the normal pancreatic duct as well as various pancreatic duct abnormalities.[10]

Hekimoglu K et al did a study to compare the diagnostic potential of one of the new MR sequences in magnetic resonance cholangiopancreatography (MRCP) procedure and endoscopic retrograde cholangiopancreatography (ERCP) with review of current literatures. They concluded that MRCP is used with increasing frequency as a non-invasive alternative to ERCP and the diagnostic results of MRCP with a heavily T2-weighted TSE MR sequence and ERCP are comparable with high accuracy in various hepatobiliary pathologies.[11]

Calvo MM et al did a study to prospectively determine the diagnostic accuracy of magnetic resonance cholangiopancreatography (MRCP) in the evaluation of the normal and diseased pancreatic duct. Patients seen during a 6-month period with a diagnosis of biliary tract or

pancreatic disease underwent endoscopic retrograde cholangiopancreatography (ERCP) after a previous MRCP. The pancreatic duct was evaluated with both techniques in 37 patients. They concluded that MRCP is an accurate technique for evaluating the normal or diseased pancreatic duct and for determining the underlying disease.[12]

Lomanto D et al did a study to assess the feasibility of MRCP versus ERCP in the diagnosis of biliary tract and pancreatic diseases. The MRCP was performed with a 0.5T superconducting magnet. When neoplastic disease was detected, additional images on axial planes were acquired. MRCP allowed images of diagnostic value to be obtained in all the cases. They concluded that MRCP can be considered a technique able to completely replace diagnostic ERCP. Further studies are necessary for a better evaluation of the potential advantages and disadvantages of this technique.[13]

Schulte B et al did a study to evaluate the accuracy of MR cholangiopancreatography (MRCP) in comparison with diagnostic findings in endoscopic retrograde cholangiopancreatography (ERCP).They concluded that even if MRCP will not replace ERCP, a number of clinical applications for non-invasive MRCP examination arise like primary diagnosis in patients with obstructive jaundice, obstruction of the biliary or pancreatic duct, if ERCP is not possible due to anatomic reason and in patients scheduled for laparoscopic cholecystectomy.[14]

Benoit P. Gallix et al did a study to evaluate the accuracy of signal intensity (SI) analysis on unenhanced fat-suppressed T1-weighted MR images in the diagnosis of pancreatic disease and to compare subjective interpretation with different quantitative measurements. They concluded that the pancreas–liver ratio is the best quantitative means of distinguishing normal from abnormal pancreas. Visual observation by experienced observers (qualitative measurement) was just as accurate as quantitative measurement. Detection of pancreatic pathology can be made with high accuracy by visually comparing the SI of the pancreas with that of the normal liver.[15]

Xiao-Ming Zhang et al did a study to assess whether measuring the pattern of pancreatic enhancement on gadolinium chelate dynamic magnetic resonance imaging (MRI) is helpful for diagnosis of suspected early or mild chronic pancreatitis. In this retrospective study, 24 patients with suspected early or mild chronic pancreatitis, classified by imaging criteria of equivocal chronic pancreatitis grading, had dynamic MRI that included unenhanced, arterial dominant, early venous, and late venous phases of contrast enhancement. They concluded that measuring pancreatic signal intensity on gadolinium chelate dynamic MRI is helpful for diagnosing early or mild chronic pancreatitis, especially before apparent pancreatic morphologic or signal intensity changes are present.

Mitchell DG et al did a study showing that Liver and pancreas: with improved spin-echo T1 contrast by shorter echo time and fat suppression at 1.5 T reported that fat saturation or water stimulation accentuates the high SI of the pancreas compared with other abdominal organs by expanding the dynamic range of intraabdominal SI and amplifying the slightly higher SI of the pancreas.[16]

Although contrast material-enhanced fat-suppressed T1-weighted images are very valuable for pancreatic carcinoma evaluation and in differentiating chronic pancreatitis from carcinoma, the comprehensive combination of pancreatic parenchymal evaluation with the use of breathhold unenhanced fat-suppressed T1-weighted images and MRCP offers excellent potential to depict pancreatic lesions solely on the basis of pancreatic duct morphology and T1-weighted SI.[17]

Temel Tirkes et all did a study to determine if the T1 relaxation time of the pancreas can detect parenchymal changes in mild chronic pancreatitis (CP). T1 maps were obtained in all patients using the same 3D gradient echo technique on the same 3T scanner. T1 relaxation times, fat signal fraction (FSF), and anterior–posterior (AP) diameter were correlated with the clinical diagnosis of CP. The T1 relaxation time of the pancreatic parenchyma was significantly increased in patients with mild CP. Therefore, T1 mapping might be

used as a practical quantitative imaging technique for the evaluation of suspected mild CP.[18]

Mougenel JL did a study to assess the accuracy and reproducibility of a new magnetic resonance cholangiopancreatography sequence (MRCP), using long echo time and "single shot" acquisition (providing high-contrast thick slices: 20 mm or more), in the morphological analysis of the biliary tree and pancreatic ducts. They concluded the excellent sensitivity, specificity and inter-observer agreement of MRCP "single shot" thick slices can be used to limit invasive imaging methods in the diagnosis of extrahepatic cholestasis.[19]

Zarine k shah et al did a study on Pancreatic parenchymal T1signal and enhancement ratio.—The normal parenchyma of the pancreas has high signal on fat-suppressed T1-weighted images, which is attributed to the higher amount of aqueous protein in the glandular elements, abundance of endoplasmic reticulum of the protein-producing acinar cells and the presence of paramagnetic ions (eg, manganese). There are several published reports of the T1-weighted signal intensity of the pancreas for CP .Some of these studies demonstrated the correlation of the T1-weighted signal intensity with pancreatic exocrine function measured with the endoscopic pancreatic function tests .These studies used T1-weighted signal intensity ratio of the pancreas to the spleen, paraspinal muscle, and the liver, all of which have been reported as useful methods for the diagnosis of CP.

Currently, there is no study comparing these methods to determine which reference organ is superior to the others. Additionally, there are instances in which the liver or spleen may have abnormal signal intensity due to iron deposition or other pathologic condition. In the absence of evidence, ratios from all reference organs are encouraged.[20]

6.3 AIMS AND OBJECTIVES

1. To study the morphological, ductal & signal intensity pattern changes of pancreas in patients with chronic pancreatitis.

7.0 MATERIALS & METHODS:

All patients who are diagnosed or suspected to have chronic pancreatitis and are referred for MRI abdomen to the Department of Radiodiagnosis, DMCH Ludhiana for a period of one and half year from 1st Dec 2019 to 31st may 2021. The diagnosed patients will be assessed for morphological changes with emphasis on signal intensity pattern and ductal changes in chronic pancreatitis. The changes in the signal intensities& pancreatic duct will be recorded in the attached proforma and the findings will be compared for better evaluation of chronic pancreatitis.

7.1 Source of Data

The study will be conducted on patients with clinical diagnosis or suspicion of CP referred from indoor or outdoor patient department to the department of Radiodiagnosis for MRI Abdomen/MRCP. The signal intensity pattern of the patients will be compared with an age and sex matched controls to define the range in patients and healthy subgroups.

7.2 METHOD OF COLLECTION OF DATA

All patients who are diagnosed to have chronic pancreatitis and are referred for MRI abdomen to the Department of Radiodiagnosis, DMCH Ludhiana for a period of 1 year. The data will be collected by personal interviews / Case Registration file / Investigations done on routine basis. Details of present and past medical history, age, will be taken into consideration followed by MR evaluation. The diagnosed patients will be assessed for morphological changes with emphasis on signal intensity pattern and ductal changes in chronic pancreatitis. The changes in the signal intensities& pancreatic duct will be recorded in the attached proforma and the findings will be compared for better evaluation of chronic pancreatitis. The various parameters of the pancreatic duct to be followed are Main pancreatic duct caliber, Pancreatic duct side branches, Pancreatic duct contour, Pancreatic duct strictures, Intraductal calculus and other parameter will include Pancreatic parenchymal T1 signal intensity, which will be compared to various reference organs as spleen, paraspinal muscle, liver and signal

	T1 vibe Fat sat	T1 vibe In –out phase	T2 haste fat sat	T2 haste traction	T2 haste coronal	T2 haste thick slab	Diffusion
TR	3.31 ms	4.34 ms	2000ms	2000ms	2000ms	4500ms	5700ms
TE	1.3 ms	1.34,2.76 mm	91ms	91ms	91ms	734ms	48ms
FOV (R)	380 mm	380 mm	350 mm	360 mm	350 mm	260 mm	380 mm
FOV (P)	81.3%	81.3%	78.1%	78.1%	100%	100%	80.6%
SLICE	72	72	30	30	35	1	30
SLICE THICK	3mm	3mm	5mm	5mm	5mm	50mm	5mm
GAP	1.1 mm	1.1mm	2.5mm	2.5mm	0.5mm	2.5mm	2.5mm
FLIP ANGLE	9^0	9^0	160^0	160^0	160^0	180^0	
FAT SAT	SPAIR	NONE	SPAIR	NONE	NONE	FATSAT	SPAIR
MATRIX	182*320	182*320	200*320	200*320	288*320	314*448	108*134
FLOW COMP	NO	NO	NO	NO	NO	NO	
PHASE ENC	AP	AP	AP	AP	R-L	R-L	AP
ORIENTATION	Transverse	Transverse	transverse	transverse	coronal		Transverse

intensity ratio of pancreas to reference organ will be calculated and a cut off value will be used to assess the parenchymal changes in chronic pancreatitis. The various MR sequences to be followed are tabulated as

Inclusion criteria:

1)Clinically diagnosed patients of chronic pancreatitis.

2)Patients with biochemical, alternative imaging and other clinical parameters suggestive of chronic pancreatitis.

Exclusion Criteria:

1) If the patient refuses to participate in the study.

2)Contraindications to MRI as per the checklist on the departmental consent form.

3)Acute pancreatitis.

4)Known cystic or solid pancreatic neoplasm.

5)Incomplete or suboptimal quality of MRI examinations.

Duration of study: One and Half year from 1st Dec 2019 to 31st May 2021.

Outcome: 1. Study will focus on various ductal changes and parenchymal signal intensity values as well as ratios of pancreas to reference organ.

Statistical analysis: 1) The application and validity of the data and the findings will be subjected to various tests of the biostatistics.

2)Data will be described in terms of range; mean +/-SD, median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. A probability value (p value) <0.05 will be considered statistically significant.

3)All statistical calculations will be done using SPSS (statistical package for the social science)SPSS 21 version statistical program for Microsoft windows. The probability value will be calculated for each of the MR parameter in predicting the mode of treatment and the treatment response.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

NO

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Attached herewith

LIST OF REFERENCES

1. Vellet AD, Romano W, Bach DB, Passi RB, Taves DH, Munk PL. Adenocarcinoma of the pancreatic ducts: comparative evaluation with CT and MR imaging at 1.5 T. Radiology. 1992;183:87–95.
2. Clain JE, Pearson RK. Diagnosis of chronic pancreatitis: is a gold standard necessary? Surg Clin North Am. 1999;79:829–45.
3. Ammann RW, Muellhaupt B. Progression of alcoholic acute to chronic pancreatitis. Gut. 1994;35:552–6.
4. Tirkes T, Shah ZK, Takahashi N, Grajo JR, Chang ST, Venkatesh SK, et al. Reporting Standards for Chronic Pancreatitis by Using CT, MRI, and MR Cholangiopancreatography: The Consortium for the Study of Chronic Pancreatitis, Diabetes and Pancreatic Cancer. Radiology. 2019;290:207-15.
5. Conwell DL, Lee LS, Yadav D, Longnecker DS, Miller FH, Mortele KJ, et al. American Pancreatic Association Practice Guidelines in Chronic Pancreatitis: evidence-based report on diagnostic guidelines. Pancreas. 2014;43:1143-62.
6. Dimcevski G, Erchinger FG, Havre R, Gilja OH. Ultrasonography in diagnosing chronic pancreatitis: new aspects. World J Gastroenterol. 2013;19:7247-57.
7. Engjom T, Kavaliauskiene G, Tjora E, Erchinger F, Wathle G, Lærum BN, et al. Sonographic pancreas echogenicity in cystic fbrosis compared to exocrine pancreatic function and pancreas fat content at Dixon-MRI. PLoS One. 2018;13:e0201019.
8. Johnson PT, Outwater EK. Pancreatic carcinoma versus chronic pancreatitis: dynamic MR imaging. Radiology. 1999;212:213–8.
9. Mitchell DG, Winston CB, Outwater EK, Ehrlich SM. Delineation of pancreas with MR imaging: multiobserver comparison of five pulse sequences. J Magn Reson Imaging. 1995;5:193–9.
10. Soto JA, Barish MA, Yucel EK, Clarke P, Siegenberg D, Chuttani R, et al. Pancreatic duct: MR cholangiopancreatography with a three-dimensional fast spin-echo technique. Radiology. 1995;196:459-64.

11. Hekimoglu K, Ustundag Y, Dusak A, Erdem Z, Karademir B, Aydemir S, et al. MRCP vs. ERCP in the evaluation of biliary pathologies: review of current literature. J Dig Dis. 2008;9:162-9.
12. Calvo MM, Calderón A, Heras I, Durán M, Orive V, Cabriada J, et al. Magnetic resonance study of the pancreatic duct. Rev Esp Enferm Dig. 1999;91:287-96.
13. Lomanto D, Pavone P, Laghi A, Panebianco V, Mazzocchi P, Fiocca F, et al. Magnetic resonance-cholangiopancreatography in the diagnosis of biliopancreatic diseases. Am J Surg. 1997;174:33-8.
14. Schulte B, Beyer D, Wedekind G, Meuser W. Single shot MRI cholangiopancreatography (MRCP) with a "fast acquisition spin echo" sequence (FASE). Replacement of ERCP? Aktuelle Radiol. 1998;8:18-24.
15. Gallix BP, Bret PM, Atri M, Lecesne R, Reinhold C. Comparison of Qualitative and Quantitative Measurements on Unenhanced T1-Weighted Fat Saturation MR Images in Predicting Pancreatic. J Magn Reson Imaging. 2005;21:583-9.
16. Tscholakoff D, Hricak H, Thoeni R, Winkler ML, Margulis AR. MR imaging in the diagnosis of pancreatic disease. AJR Am J Roentgenol 1987;148:703–709.
17. Matos C, Nicaise N, Metens T. Secretin-enhanced MR pancreatography. Semin Ultrasound CT MR. 1999;20:340–351
18. Kimelman T, Vu A, Storey P, McKenzie C, Burstein D, Prasad P. Three-dimensional T1 mapping for dGEMRIC at 3.0 T using the Look Locker method. Investig Radiol. 2006;41:198–203.
19. Mougenel JL, Hudziak H, Ernst O, Dupas B, Lefevre F, Barraud H, et al. Evaluation of a new sequence of magnetic resonance cholangio-pancreatography in thick cut and one shot acquisition]. Gastroenterol Clin Biol. 2000;24:888-95.
20. Winston CB, Mitchell DG, Outwater EK, Ehrlich SM. Pancreatic signal intensity on T1-weighted fat saturation MR images: clinical correlation. J Magn Reson Imaging 1995;5:267–71.

PROFORMA

Sr. No.

Patient's Name: Occupation:

Date of requisition : MRD No. :

Age of patient : Contact No. :

Address :

Consultant :

History of present illness:

Past medical history:

Examination findings:

Investigations:

Hb- TLC- Bilirubin [T/D]-

SGOT- SGPT- ALP-

S. Creatinine-

Special investigations:

S. Amylase- S. Lipase- Lipid profile-

S.trypsinogen- Fecal elastase-

Signature of Candidate **Signature of Supervisor**

Previous radiological (USG, CT or MR abdomen), endoscopy studies-

Current MRI / MRCP findings-

1. Main pancreatic duct caliber
2. No of pancreatic duct side branches
3. Pancreatic duct contour
4. Location of Pancreatic duct strictures
5. Intraductal calculus
6. Pancreatic parenchymal T1 signal intensity
7. T1 signal intensity ratio($SIR_{p/ro}$)
8. Pancreatic parenchymal thickness
9. Distribution of findings

Follow up of the patient

Signature of Candidate **Signature of Supervisor**

INFORMED PATIENT CONSENT

Date: _____

I.....D/o, W/o, S/o..... .....am willing to participate in the study **"ROLE OF MAGNETIC RESONANCE IMAGING IN CHRONIC PANCREATITIS"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

....

Signature of the Witness Signature of the patient

Name:

Signature of Candidate **Signature of Supervisor**

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य **"ROLE OF MAGNETIC RESONANCE IMAGING IN CHRONIC PANCREATITIS"** में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

"ROLE OF MAGNETIC RESONANCE IMAGING IN CHRONIC PANCREATITIS" ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

_____ _____

ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ _____ ਪਤਾ _____

_____

ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

_____

ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ